10/045 631

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=> d his

     (FILE 'HOME' ENTERED AT 08:59:27 ON 17 SEP 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 08:59:47 ON 17 SEP 2004
L1         14477 S KINESIN?
L2           831 S "CENP-E"
L3             1 S "CENTROMER BINDING"
L4             0 S CENTROMER (2W) "PROTEIN E"
L5           282 S L1 AND L2
L6           125 S HUMAN AND L5
L7            67 S MOTOR AND L6
L8        333307 S ATPASE
L9             6 S L6 AND L8
L10            6 DUP REM L9 (0 DUPLICATES REMOVED)
L11           30 DUP REM L7 (37 DUPLICATES REMOVED)
                 E BEARUD C/AU
                 E BERAUD C/AU
L12          478 S E3
                 E OHASHI C/AU
L13           26 S E3
                 E SAKOWICZ R/AU
L14           76 S E5
                 E VAISBERG E/AU
L15           30 S E3
                 E WOOD K/AU
L16          803 S E3
                 E YU M/AU
L17         2350 S E3
L18         3786 S L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17
L19           36 S L2 AND L18
L20           34 DUP REM L19 (2 DUPLICATES REMOVED)

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NEWS 3	May 12	EXTEND option available in structure searching
NEWS 4	May 12	Polymer links for the POLYLINK command completed in REGISTRY
NEWS 5	May 27	New UPM (Update Code Maximum) field for more efficient patent SDIs in CAplus
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NEWS 10	Jul 30	BEILSTEIN on STN workshop to be held August 24 in conjunction with the 228th ACS National Meeting
NEWS 11	AUG 02	IFIPAT/IFIUDB/IFICDB reloaded with new search and display fields
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NEWS 16	AUG 27	BIOCOMMERCE: Changes and enhancements to content coverage
NEWS 17	AUG 27	BIOTECHABS/BIOTECHDS: Two new display fields added for legal status data from INPADOC
NEWS 18	SEP 01	INPADOC: New family current-awareness alert (SDI) available
NEWS 19	SEP 01	New pricing for the Save Answers for SciFinder Wizard within STN Express with Discover!
NEWS 20	SEP 01	New display format, HITSTR, available in WPIDS/WPINDEX/WPIX
NEWS 21	SEP 14	STN Patent Forum to be held October 13, 2004, in Iselin, NJ

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FILE 'HOME' ENTERED AT 08:59:27 ON 17 SEP 2004

=> file medline embase biosis biotechds scisearch hcaplus ntis lifesci
COST IN U.S. DOLLARS SINCE FILE ENTRY TOTAL SESSION
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FILE 'MEDLINE' ENTERED AT 08:59:47 ON 17 SEP 2004

FILE 'EMBASE' ENTERED AT 08:59:47 ON 17 SEP 2004

FILE 'BIOSIS' ENTERED AT 08:59:47 ON 17 SEP 2004

FILE 'BIOTECHDS' ENTERED AT 08:59:47 ON 17 SEP 2004

FILE 'SCISEARCH' ENTERED AT 08:59:47 ON 17 SEP 2004

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FILE 'LIFESCI' ENTERED AT 08:59:47 ON 17 SEP 2004

=> s kinesin?
L1 14477 KINESIN?

=> s "CENP-E"
L2 831 "CENP-E"

=> s "centromer binding"
L3 1 "CENTROMER BINDING"

=> s centromer (2w) "protein E"
L4 0 CENTROMER (2W) "PROTEIN E"

=> s l1 and l2
L5 282 L1 AND L2

=> s human and l5
L6 125 HUMAN AND L5

=> s motor and l6
L7 67 MOTOR AND L6

=> s ATPase
L8 333307 ATPASE

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=> s l6 and l8
L9            6 L6 AND L8

=> dup rem l9
PROCESSING COMPLETED FOR L9
L10           6 DUP REM L9 (0 DUPLICATES REMOVED)

=> d 1-6 ibib ab

L10  ANSWER 1 OF 6  HCAPLUS  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:         2003:409169  HCAPLUS
DOCUMENT NUMBER:          138:380506
TITLE:                    Genes that are differentially expressed during
                          erythropoiesis and their diagnostic and therapeutic
                          uses
INVENTOR(S):              Brissette, William H.; Neote, Kuldeep S.; Zagouras,
                          Panayiotis; Zenke, Martin; Lemke, Britt; Hacker,
                          Christine
PATENT ASSIGNEE(S):       Pfizer Products Inc., USA; Max-Delbrueck-Centrum Fuer
                          Molekulare Medizin
SOURCE:                   PCT Int. Appl., 285 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   2
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.        DATE
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     WO 2003038130         A2    20030508    WO 2002-XA34888        20021031
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,
             TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR,
             NE, SN, TD, TG
     WO 2003038130         A2    20030508    WO 2002-US34888        20021031
     WO 2003038130         A3    20040212
     WO 2003038130         C1    20040422
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU,
             TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR,
             NE, SN, TD, TG
PRIORITY APPLN. INFO.:                    US 2001-335048P     P  20011031
                                          US 2001-335183P     P  20011102
                                          WO 2002-US34888     A  20021031
AB   The present invention provides mol. targets that regulate erythropoiesis.
     Groups of genes or their encoded gene products comprise panels of the
     invention and may be used in therapeutic intervention, therapeutic agent
     screening, and in diagnostic methods for diseases and/or disorders of
     erythropoiesis.  The panels were discovered using gene expression
     profiling of erythroid progenitors with Affymetrix HU6800 and HG-U95Av2
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chips. Cells from an in vitro growth and differentiation system of SCF-Epo dependent **human** erythroid progenitors, E-cadherin+/CD36+ progenitors, cord blood, or CD34+ peripheral blood stem cells were analyzed. The HU6800 chip contains probes from 13,000 genes with a potential role in cell growth, proliferation, and differentiation and the HG-U95Av2 chip contains 12,000 full-length, functionally-characterized genes. [This abstract record is one of two records for this document necessitated by the large number of index entries required to fully index the document and publication system constraints.].

L10 ANSWER 2 OF 6 MEDLINE on STN
ACCESSION NUMBER: 2001338615 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11250166
TITLE: Chromosome movement: dynein-out at the kinetochore.
AUTHOR: Banks J D; Heald R
CORPORATE SOURCE: Department of Molecular and Cell Biology, University of California, Berkeley, California 94720-3200, USA.. jenbanks@uclink4.berkeley.edu
SOURCE: Current biology : CB, (2001 Feb 20) 11 (4) R128-31. Ref: 28
Journal code: 9107782. ISSN: 0960-9822.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010618
Last Updated on STN: 20010618
Entered Medline: 20010614

AB Cell biologists have long speculated that a minus end-directed motor localized at kinetochores contributes to the poleward movement of chromosomes during mitosis. Two recent studies provide direct evidence that cytoplasmic dynein can perform this function.

L10 ANSWER 3 OF 6 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:756837 HCAPLUS
DOCUMENT NUMBER: 133:318271
TITLE: Recombinant bacterial expression and purification of **human kinesins**
INVENTOR(S): Beraud, Christophe; Ohashi, Cara; Sakowicz, Roman; Wood, Ken; Vaisberg, Eugeni; Yu, Ming
PATENT ASSIGNEE(S): Cytokinetics, USA
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000063353	A1	20001026	WO 2000-US10870	20000420
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6544766	B1	20030408	US 2000-595684	20000616

US 6387644	B1	20020514	US 2000-724224	20001128
US 2003044900	A1	20030306	US 2001-45631	20011019
US 6762043	B1	20040713	US 2002-93317	20020306
US 2004142397	A1	20040722	US 2004-797893	20040309

PRIORITY APPLN. INFO.: US 1999-295612 A1 19990420
WO 2000-US10870 A1 20000420
US 2000-597292 B1 20000620
US 2000-724224 A1 20001128
US 2002-93317 A3 20020306

AB Described herein are methods of producing **kinesins**. In a preferred embodiment, the **kinesins** are produced from a prokaryote, most preferably, a bacterial cell. Bacterial expression offers several advantages over systems previously utilized, such as, for example, Baculovirus. The yield of protein is higher, the cost of the expression setup is lower, and creation of alternative expression vectors is easier. The concern of copurifying a contaminating activity from the expression host is also eliminated since bacteria, in contrast to the baculovirus expression system, do not have **kinesin**-like proteins. Also described herein are purified **kinesins**, preferably unglycosylated and methods of use.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 4 OF 6 MEDLINE on STN
ACCESSION NUMBER: 1998167852 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9499420
TITLE: Localization of motor-related proteins and associated complexes to active, but not inactive, centromeres.
AUTHOR: Faulkner N E; Vig B; Echeverri C J; Wordeman L; Vallee R B
CORPORATE SOURCE: Cell Biology Group, Worcester Foundation for Biomedical Research, Shrewsbury, MA 01545, USA.
CONTRACT NUMBER: GM478434 (NIGMS)
SOURCE: Human molecular genetics, (1998 Apr) 7 (4) 671-7.
Journal code: 9208958. ISSN: 0964-6906.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980520
Last Updated on STN: 19980520
Entered Medline: 19980512

AB Multicentric chromosomes are often found in tumor cells and certain cell lines. How they are generated is not fully understood, though their stability suggests that they are non-functional during chromosome segregation. Growing evidence has implicated microtubule motor proteins in attachment of chromosomes to the mitotic spindle and in chromosome movement. To better understand the molecular basis for the inactivity of centromeres associated with secondary constrictions, we have tested these structures by immunofluorescence microscopy for the presence of motor complexes and associated proteins. We find strong immunoreactivity at the active, but not inactive, centromeres of prometaphase multicentric chromosomes using antibodies to the cytoplasmic dynein intermediate chains, three components of the dynactin complex (dynamitin, Arp1 and p150 Glued), the **kinesin**-related proteins **CENP-E** and MCAK and the proposed structural and checkpoint proteins HZW10, CENP-F and Mad2p. These results offer new insight into the assembly and composition of both primary and secondary constrictions and provide a molecular basis for the apparent inactivity of the latter during chromosome segregation.

L10 ANSWER 5 OF 6 MEDLINE on STN
ACCESSION NUMBER: 95196755 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7889940

TITLE: Mitotic HeLa cells contain a **CENP-E** -associated minus end-directed microtubule motor.
AUTHOR: Thrower D A; Jordan M A; Schaar B T; Yen T J; Wilson L
CORPORATE SOURCE: Department of Biological Sciences, University of California, Santa Barbara 93106.
CONTRACT NUMBER: CA06927 (NCI)
GM44762 (NIGMS)
SOURCE: EMBO journal, (1995 Mar 1) 14 (5) 918-26.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 19950427
Last Updated on STN: 19970203
Entered Medline: 19950420

AB A minus end-directed microtubule motor activity from extracts of HeLa cells blocked at prometaphase/metaphase of mitosis with vinblastine has been partially purified and characterized. The motor activity was eliminated by immunodepletion of Centromere binding protein E (**CENP-E**). The **CENP-E**-associated motor activity, which was not detectable in interphase cells, moved microtubules at mean rates of 0.46 micron/s at 37 degrees C and 0.24 micron/s at 25 degrees C. The motor activity co-purified with **CENP-E** through several purification procedures. Motor activity was clearly not due to dynein or to **kinesin**. The microtubule gliding rates of the **CENP-E**-associated motor were different from those of dynein and **kinesin**. In addition, the pattern of nucleotide substrate utilization by the **CENP-E**-associated motor and the sensitivity to inhibitors were different from those of dynein and **kinesin**. The **CENP-E**-associated motor had an apparent native molecular weight of 874,000 Da and estimated dimensions of 2 nm x 80 nm. This is the first demonstration of motor activity associated with **CENP-E**, strongly supporting the hypothesis that **CENP-E** may act as a minus end-directed microtubule motor during mitosis.

L10 ANSWER 6 OF 6 MEDLINE on STN
ACCESSION NUMBER: 95122643 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7822426
TITLE: Identification and partial characterization of mitotic centromere-associated **kinesin**, a **kinesin** -related protein that associates with centromeres during mitosis.
COMMENT: Comment in: J Cell Biol. 1995 Jan;128(1-2):1-4. PubMed ID: 7822407
AUTHOR: Wordeman L; Mitchison T J
CORPORATE SOURCE: Department of Physiology and Biophysics, University of Washington, Seattle 98195.
CONTRACT NUMBER: CA-09270 (NCI)
GM-39565 (NIGMS)
SOURCE: Journal of cell biology, (1995 Jan) 128 (1-2) 95-104.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950223
Last Updated on STN: 20021227
Entered Medline: 19950214

AB Using antipeptide antibodies to conserved regions of the **kinesin**

motor domain, we cloned a **kinesin**-related protein that associates with the centromere region of mitotic chromosomes. We call the protein MCAK, for mitotic centromere-associated **kinesin**. MCAK appears concentrated on centromeres at prophase and persists until telophase, after which time the localization disperses. It is found throughout the centromere region and between the kinetochore plates of isolated mitotic CHO chromosomes, in contrast to two other kinetochore-associated microtubule motors: cytoplasmic dynein and **CENP-E** (Yen et al., 1992), which are closer to the outer surface of the kinetochore plates. Sequence analysis shows MCAK to be a **kinesin**-related protein with the motor domain located in the center of the protein. It is 60-70% similar to kif2, a **kinesin** -related protein originally cloned from mouse brain with a centrally located motor domain (Aizawa et al., 1992). MCAK protein is present in interphase and mitotic CHO cells and is transcribed as a single 3.4-kb message.

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     (FILE 'HOME' ENTERED AT 08:59:27 ON 17 SEP 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 08:59:47 ON 17 SEP 2004
L1         14477 S KINESIN?
L2           831 S "CENP-E"
L3             1 S "CENTROMER BINDING"
L4             0 S CENTROMER (2W) "PROTEIN E"
L5           282 S L1 AND L2
L6           125 S HUMAN AND L5
L7            67 S MOTOR AND L6
L8        333307 S ATPASE
L9             6 S L6 AND L8
L10            6 DUP REM L9 (0 DUPLICATES REMOVED)

=> dup rem l7
PROCESSING COMPLETED FOR L7
L11           30 DUP REM L7 (37 DUPLICATES REMOVED)

=> d 1-30 ibib ab

L11  ANSWER 1 OF 30  EMBASE  COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN                                              DUPLICATE 1
ACCESSION NUMBER:   2004325682  EMBASE
TITLE:              Gene silencing of CENP-E by small
                    interfering RNA in HeLa cells leads to missegregation of
                    chromosomes after a mitotic delay.
AUTHOR:             Tanudji M.; Shoemaker J.; L'Italien L.; Russell L.; Chin
                    G.; Schebye X.M.
CORPORATE SOURCE:   X.M. Schebye, DNAX Research Institute, Palo Alto, CA 94304,
                    United States. xiaomin.schebye@dnax.org
SOURCE:             Molecular Biology of the Cell, (2004) 15/8 (3771-3781).
                    Refs: 33
                    ISSN: 1059-1524  CODEN: MBCEEV
COUNTRY:            United States
DOCUMENT TYPE:      Journal; Article
FILE SEGMENT:       029     Clinical Biochemistry
LANGUAGE:           English
SUMMARY LANGUAGE:   English
AB   Centromeric protein-E (CENP-E) is a kinesin
     -like motor protein required for chromosome congression at
     prometaphase. Functional perturbation of CENP-E by
     various methods results in a consistent phenotype, i.e., unaligned
     chromosomes during mitosis. One unresolved question from previous studies
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is whether cells complete mitosis or sustain mitotic arrest in the presence of unaligned chromosomes. Using RNA interference and video-microscopy, we analyzed the dynamic process of mitotic progression of HeLa(H2B)-GFP cells lacking **CENP-E**. Our results demonstrate that these cells initiated anaphase after a delayed mitotic progression due to the presence of unaligned chromosomes. In some dividing cells, unaligned chromosomes are present during anaphase, causing nondisjunction of some sister chromatids producing aneuploid daughter cells. Unlike in Xenopus extract, the loss of **CENP-E** in HeLa cells does not impair gross checkpoint activation because cells were arrested in mitosis in response to microtubule-interfering agents. However, the lack of **CENP-E** at kinetochores reduced the hyperphosphorylation of BubR1 checkpoint protein during mitosis, which may explain the loss of sensitivity of a cell to a few unaligned chromosomes in the absence of **CENP-E**. We also found that presynchronization with nocodazole sensitizes cells to the depletion of **CENP-E**, leading to more unaligned chromosomes, longer arrest, and cell death.

L11 ANSWER 2 OF 30 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2004258559 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 15159587
TITLE: Crystallization and preliminary crystallographic analysis of the **motor** domain of **human** kinetochore-associated protein **CENP-E** using an automated crystallization procedure.
AUTHOR: Garcia-Saez Isabel; Blot Delphine; Kahn Richard; Kozielski Frank
CORPORATE SOURCE: Laboratoire de Microscopie Electronique Structurale, Institut de Biologie Structurale Jean-Pierre Ebel (CEA-CNRS-UJF), 41 Rue Jules Horowitz, 38027 Grenoble CEDEX 01, France.. isabel.garcia@ibs.fr
SOURCE: Acta crystallographica. Section D, Biological crystallography, (2004 Jun) 60 (Pt 6) 1158-60. Journal code: 9305878. ISSN: 0907-4449.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20040526
Last Updated on STN: 20040629
AB **Human** centromere-associated protein E, a member of the **kinesin** superfamily, is a microtubule-dependent **motor** protein involved in cell division that has been localized transiently to the kinetochore. The protein is thought to be responsible for the correct attachment and positioning of chromosomes to the mitotic spindle during the metaphase. The 312 kDa protein comprises four different domains. In this study, the focus was on the N-terminal **motor** domain, which includes the ATP-binding site and a region for microtubule binding. Crystals of the **CENP-E motor** domain have been obtained by high-throughput crystallization screening using an automated TECAN crystallization robot. The crystals (737 x 132 x 79 microm) belong to the space group P2(1), with unit-cell parameters a = 49.35, b = 83.70, c = 94.16 angstroms, beta = 103.05 degrees. They diffract to 2.1 angstroms resolution using synchrotron radiation.

L11 ANSWER 3 OF 30 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2004334945 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15236970
TITLE: Crystal structure of the **motor** domain of the **human** kinetochore protein **CENP-E**
.
AUTHOR: Garcia-Saez Isabel; Yen Tim; Wade Richard H; Kozielski

Frank
CORPORATE SOURCE: Laboratoire de Microscopie Electronique Structurale, Institut de Biologie Structurale, 41 rue Jules Horowitz, 38027 Grenoble Cedex 01, France.
CONTRACT NUMBER: CA06927 (NCI)
CA75138 (NCI)
GM44762 (NIGMS)
SOURCE: Journal of molecular biology, (2004 Jul 23) 340 (5) 1107-16.
Journal code: 2985088R. ISSN: 0022-2836.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200408
ENTRY DATE: Entered STN: 20040707
Last Updated on STN: 20040826
Entered Medline: 20040825
AB The **human** kinetochore is a highly complex macromolecular structure that connects chromosomes to spindle microtubules (MTs) in order to facilitate accurate chromosome segregation. Centromere-associated protein E (**CENP-E**), a member of the **kinesin** superfamily, is an essential component of the kinetochore, since it is required to stabilize the attachment of chromosomes to spindle MTs, to develop tension across aligned chromosomes, to stabilize spindle poles and to satisfy the mitotic checkpoint. Here we report the 2.5A resolution crystal structure of the **motor** domain and linker region of **human CENP-E** with MgADP bound in the active site. This structure displays subtle but important differences compared to the structures of **human** Eg5 and conventional **kinesin** . Our structure reveals that the **CENP-E** linker region is in a "docked" position identical to that in the **human** plus-end directed conventional **kinesin**. **CENP-E** has many advantages as a potential anti-mitotic drug target and this crystal structure of **human CENP-E** will provide a starting point for high throughput virtual screening of potential inhibitors.

L11 ANSWER 4 OF 30 BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP/ISI on STN
ACCESSION NUMBER: 2003-15387 BIOTECHDS
TITLE: Treatment of disease e.g. cancer, rheumatoid arthritis, Alzheimer's disease and Parkinson's disease involves administration of antisense oligonucleotide; **human kinesin**-specific oligonucleotide transfer and expression in host cell for gene therapy
AUTHOR: REINHARD C; WALTER A
PATENT ASSIGNEE: CHIRON CORP
PATENT INFO: WO 2003030832 17 Apr 2003
APPLICATION INFO: WO 2002-US32596 11 Oct 2002
PRIORITY INFO: US 2001-328444 12 Oct 2001; US 2001-328444 12 Oct 2001
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-381676 [36]
AB DERWENT ABSTRACT:
NOVELTY - Treatment of disease involves administering an antisense oligonucleotide. The oligonucleotide inhibits the expression of **human kinesin** gene. The **human kinesin** gene is **CENP-E**, **human** Eg5 or MCAK.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following: (1) an anti-sense oligonucleotide (I) having nucleic acid sequence CCTCCGCCATCCTATCAGGCTGAA, CCGAGGAGAAAGCGAAATAGGGAAG, GAGACCGACTCTTGCTCTGTTGCC, GTTGATCTGGGCTCGCAGAGGTAAT, CTCTGTGGTGTCGTACCTGTTGGGA, TGGGTTCAAGTGATTCTCGTGCCTC, TGTCAGCCAATCCTCCAGTTCGTAC, TTGTACGCCCTCCAAGAGAATCCTG,

GCTCAAGCAATCCACCCGCCTCAG, GGGATTACAGGCATGAGCCACCGC, CACTCCATTTTTCTCACGGGCTGCA, CATTCTCCTGAGCCGTGATGCGAA, ACGGAACGGGGTGTGAGCCTTGT, TGTCAGCTTGCTCTCACGGAACGG, GGAGCTTATGCCTGGTGAGATCGTG, GAGTCAGCAAGGAAGAGAAACGCG, TGGATAAATTGCCTGGAATCAGCGC and CGTTGGATCTTGATAGCGAGACCGG (2) combination therapy involving administration of at least one chemotherapeutic or radionuclide and further involves administration of at least one anti-sense oligonucleotide, the oligonucleotide is administered either separately or in combination; and (3) a pharmaceutical composition comprising (I) and a carrier.

ACTIVITY - Cytostatic; Immunosuppressive; Virucide; Vasotropic; Cerebroprotective; Cardiant; Antibacterial; Fungicide; Protozoacide; Antirheumatic; Antiarthritic; Antiinflammatory; Anticonvulsant; Antiparkinsonian; Nootropic; Neuroprotective; Neuroleptic; CNS-Gen.; Sedative; Dermatological; Analgesic; Tranquilizer; Antidiabetic; Antilipemic; Nephrotropic; Gastrointestinal-Gen.; Antiulcer; Anti-HIV; Antiallergic; Antianemic; Osteopathic; Anthelmintic; Ophthalmological; Antithyroid; Respiratory-Gen.

MECHANISM OF ACTION - **Human kinesin** gene inhibitor; Modulator of function of nucleic acid molecule encoding **human kinesin**; Anchorage independent growth inhibitor.
The antisense oligonucleotide of sequence TGGATAAATTGCCTGGAATCAGCGC (i) was transfected into **human** colon cancer cell line SW620. The same colon cancer cell line was transfected with the corresponding reverse control sequence CGCGACTAAGGTCCGTTAAATAGGT (ii). The total number of colonies normalized were: for (i) was approximately 425 and for (ii) was approximately 800. The results showed that the antisense oligonucleotide inhibited the capability of the cells to grow in soft agar and inhibited anchorage independent growth. The results showed that the **kinesin** antisense oligonucleotide inhibited tumorigenesis.

USE - For treatment of disease having aberrant cell proliferation such as cancer e.g. colon cancer, T and B cell lymphoma, pancreatic cancer, breast cancer, leukemia, bladder cancer, stomach cancer, brain cancer, esophageal cancer, liver cancer, adrenalcarcinoma, lung cancer, testicular cancer, heart cancer, ovarian cancer, uterine cancer, head and neck cancer, bone cancer, cervical cancer, gall bladder cancer, parathrnoid cancer, penile cancer, prostate cancer, skin cancer, spleen cancer, thymus cancer, thyroid cancer, muscle cancer, ganglial cancer, melanoma, myeloma sarcoma and teratocarcinomas, digestive cancer, lymphoma, autoimmune disorder, viral infection, neurological disorder, condition associated with ischemia and liver or pancreatic disease (claimed), myocardial infarction and stroke. The neurological disorders e.g. epilepsy, ischemic cerebrovascular disease, cerebral neoplasm, Alzheimer's disease, Pick's disease, Huntington's disease, dementia, Parkinson's disease, extrapyramidal disorder, amyotrophic lateral sclerosis, **motor** neuron disorders, progressive neural muscular atrophy, retinitis pigmentosa, hereditary ataxia, suppurative intracranial thrombophlebitis, multiple sclerosis, demyelinating disease, bacterial and viral meningitis, brain abscess, subdural empyema, myelitis, paralysis, viral central nervous system disease, prion disease including kuru, Creutzfeldt-Jakob disease, Gerstmann-Straussler-Scheinker syndrome, insomnia, neurofibromatosis, mental retardation, cerebral palsy, autonomic nervous system disorder, muscular dystrophy, peripheral nervous system disorders, dermatomyositis, anxiety, schizophrenia, amnesia, diabetic neuropathy, tardive dyskinesia, Tourette's disease, cystic fibrosis, hypercholesterolemia, diabetic mellitus, hyper- and hypoglycemia, Grave's disease, neuralgia, Cushing's disease, Addison's disease, gastrointestinal disorders e.g. ulcerative colitis, duodenal ulcer, AIDS, allergic reactions, autoimmune hemolytic anemia, proliferative glomerulonephritis, inflammatory bowel disease, myasthenia gravis, rheumatoid arthritis, osteoarthritis, scleroderma, Sjogren's syndrome, systemic lupus erythematosus, toxic shock syndrome, viral, bacterial, fungal, helminthic and protozoal infections.

ADMINISTRATION - The composition is administered orally,

intranasally, anally, topically or by injection (claimed), parenterally (including intravenously, intraarterially, subcutaneously, intraperitoneally, intracranially, intramuscularly or by infusion), intrathecally, intraventricularly, locally, systemically, vaginally, rectally, pulmonary, by inhalation, as aerosol, intranasally, epidermally, transdermally, as liposome or ophthalmically in a dosage of 0.01 ug - 100 g.

ADVANTAGE - The anti-sense oligonucleotide inhibits expression of **human kinesin** gene such as **CENP-E** having nucleic acid sequence deposited in GenBank as GenBank ID Z15005, **human** Eg5 having nucleic acid sequence deposited in GenBank as GenBank ID U37426 and MCAK gene having nucleic acid sequence deposited in GenBank as GenBank ID U63743.

EXAMPLE - No relevant example given. (29 pages)

L11 ANSWER 5 OF 30 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2002:950597 SCISEARCH
THE GENUINE ARTICLE: 615PA
TITLE: The mitotic-spindle-associated protein astrin is essential for progression through mitosis
AUTHOR: Gruber J; Harborth J; Schnabel J; Weber K; Hatzfeld M (Reprint)
CORPORATE SOURCE: Univ Halle Wittenberg, Fac Med, Dept Biochem & Pathobiochem, D-06097 Halle Saale, Germany (Reprint); Max Planck Inst Biophys Chem, Dept Biochem, D-37070 Gottingen, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: JOURNAL OF CELL SCIENCE, (1 NOV 2002) Vol. 115, No. 21, pp. 4053-4059.
Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE CB4 4DL, CAMBS, ENGLAND.
ISSN: 0021-9533.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 41

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Astrin is a mitotic-spindle-associated protein expressed in most **human** cell lines and tissues. However, its functions in spindle organization and mitosis have not yet been determined. Sequence analysis revealed that astrin has an N-terminal globular domain and an extended coiled-coil domain. Recombinant astrin was purified and characterized by CD spectroscopy and electron microscopy. Astrin showed parallel dimers with head-stalk structures reminiscent of **motor** proteins, although no sequence similarities to known **motor** proteins were found. In physiological buffers, astrin dimers oligomerized via their globular head domains and formed aster-like structures. Silencing of astrin in HeLa cells by RNA interference resulted in growth arrest, with formation of multipolar and highly disordered spindles. Chromosomes did not congress to the spindle equator and remained dispersed. Cells depleted of astrin were normal during interphase but were unable to progress through mitosis and finally ended in apoptotic cell death. Possible functions of astrin in mitotic spindle organization are discussed.

L11 ANSWER 6 OF 30 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN DUPLICATE 4

ACCESSION NUMBER: 2002:978474 SCISEARCH
THE GENUINE ARTICLE: 621KX
TITLE: Protein kinase TTK interacts and co-localizes with **CENP-E** to the kinetochore of **human** cells
AUTHOR: Zhang J; Fu C H; Miao Y; Dou Z; Yao X B (Reprint)
CORPORATE SOURCE: Univ Sci & Technol China, Lab Cell Dynam, Hefei 230027,

Peoples R China (Reprint)
COUNTRY OF AUTHOR: Peoples R China
SOURCE: CHINESE SCIENCE BULLETIN, (DEC 2002) Vol. 47, No. 23, pp. 2005-2009.
Publisher: SCIENCE CHINA PRESS, 16 DONGHUANGCHENGGEN NORTH ST, BEIJING 100717, PEOPLES R CHINA.
ISSN: 1001-6538.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 25

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Spindle checkpoint is an important biochemical signaling cascade during mitosis which monitors the fidelity of chromosome segregation, and is mediated by protein kinases Mps1 and Bub1/BubR1. Our recent studies show that **kinesin**-related **motor** protein **CENP-E** interacts with BubR1 and participates in spindle checkpoint signaling. To elucidate the molecular mechanisms underlying spindle checkpoint signaling, we carried out proteomic dissection of **human** cell kinetochore and revealed protein kinase TTK, **human** homologue of yeast Mps1. Our studies show that TTK is, localized to the kinetochore of **human** cells, and interacts with **CENP-E**, suggesting that TTK may play an important role in chromosome segregation during mitosis.

L11 ANSWER 7 OF 30 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2002:174077 SCISEARCH
THE GENUINE ARTICLE: 520WH
TITLE: Zebrafish mitotic **kinesin**-like protein 1 (Mklp1) functions in embryonic cytokinesis
AUTHOR: Chen M C; Zhou Y; Detrich H W (Reprint)
CORPORATE SOURCE: Northeastern Univ, Dept Biol, 414 Mugar Hall, 360 Huntington Ave, Boston, MA 02115 USA (Reprint); Northeastern Univ, Dept Biol, Boston, MA 02115 USA; Childrens Hosp, Div Hematol Oncol, Boston, MA 02115 USA; Howard Hughes Med Inst, Boston, MA 02115 USA
COUNTRY OF AUTHOR: USA
SOURCE: PHYSIOLOGICAL GENOMICS, (11 FEB 2002) Vol. 8, No. 1, pp. 51-66.
Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 1094-8341.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 76

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB To understand the functions of microtubule **motors** in vertebrate development, we are investigating the **kinesin**-like proteins (KLPs) of the zebrafish, Danio rerio. Here we describe the structure, intracellular distribution, and function of zebrafish mitotic KLP1 (Mklp1). The zebrafish mklp1 gene that encodes this 867-amino acid protein maps to a region of zebrafish linkage group 18 that is syntenic with part of **human** chromosome 15. In zebrafish AB9 fibroblasts and in COS-7 cells, the zebrafish Mklp1 protein decorates spindle microtubules at metaphase, redistributes to the spindle midzone during anaphase, and becomes concentrated in the midbody during telophase and cytokinesis. The **motor** is detected consistently in interphase nuclei of COS cells and occasionally in those of AB9 cells. Nuclear targeting of Mklp1 is conferred by two basic motifs located in the COOH terminus of the **motor**. In cleaving zebrafish embryos, green fluorescent protein (GFP)-tagged Mklp1 is found in the nucleus in interphase and associates with microtubules of the spindle midbody in cytokinesis. One- or two-cell embryos injected with synthetic mRNAs encoding dominant-negative variants of GFP-Mklp1 frequently fail to

complete cytokinesis during cleavage, resulting in formation of multinucleated blastomeres. Our results indicate that the zebrafish Mklp1 **motor** performs a critical function that is required for completion of embryonic cytokinesis.

L11 ANSWER 8 OF 30 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2001688509 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11734897
TITLE: Maximum likelihood methods reveal conservation of function among closely related **kinesin** families.
AUTHOR: Lawrence Carolyn J; Malmberg Russell L; Muszynski Michael G; Dawe R Kelly
CORPORATE SOURCE: University of Georgia, Department of Botany, Athens, GA 30602, USA.
SOURCE: Journal of molecular evolution, (2002 Jan) 54 (1) 42-53.
Journal code: 0360051. ISSN: 0022-2844.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20011206
Last Updated on STN: 20020816
Entered Medline: 20020815

AB We have reconstructed the evolution of the anciently derived **kinesin** superfamily using various alignment and tree-building methods. In addition to classifying previously described **kinesins** from protists, fungi, and animals, we analyzed a variety of **kinesin** sequences from the plant kingdom including 12 from Zea mays and 29 from Arabidopsis thaliana. Also included in our data set were four sequences from the anciently diverged amitochondriate protist Giardia lamblia. The overall topology of the best tree we found is more likely than previously reported topologies and allows us to make the following new observations: (1) **kinesins** involved in chromosome movement including MCAK, chromokinesin, and **CENP-E** may be descended from a single ancestor; (2) **kinesins** that form complex oligomers are limited to a monophyletic group of families; (3) **kinesins** that crosslink antiparallel microtubules at the spindle midzone including BIMC, MKLP, and **CENP-E** are closely related; (4) Drosophila NOD and **human** KID group with other characterized chromokinesins; and (5) Saccharomyces SMY1 groups with **kinesin**-I sequences, forming a family of **kinesins** capable of class V myosin interactions. In addition, we found that one monophyletic clade composed exclusively of sequences with a C-terminal **motor** domain contains all known minus end-directed **kinesins**.

L11 ANSWER 9 OF 30 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2001417117 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11382767
TITLE: Purification and characterization of native conventional **kinesin**, HSET, and **CENP-E** from mitotic hela cells.
AUTHOR: DeLuca J G; Newton C N; Himes R H; Jordan M A; Wilson L
CORPORATE SOURCE: Department of Molecular, Cellular, and Developmental Biology and the Materials Research Laboratory, University of California, Santa Barbara, California 93106, USA.
CONTRACT NUMBER: CA57291 (NCI)
NS13560 (NINDS)
SOURCE: Journal of biological chemistry, (2001 Jul 27) 276 (30) 28014-21.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010827
Last Updated on STN: 20030105
Entered Medline: 20010823

AB We have developed a strategy for the purification of native microtubule **motor** proteins from mitotic HeLa cells and describe here the purification and characterization of **human** conventional **kinesin** and two **human kinesin**-related proteins, HSET and **CENP-E**. We found that the 120-kDa HeLa cell conventional **kinesin** is an active **motor** that induces microtubule gliding at approximately 30 microm/min at room temperature. This active form of HeLa cell **kinesin** does not contain light chains, although light chains were detected in other fractions. HSET, a member of the C-terminal **kinesin** subfamily, was also purified in native form for the first time, and the protein migrates as a single band at approximately 75 kDa. The purified HSET is an active **motor** that induces microtubule gliding at a rate of approximately 5 microm/min, and microtubules glide for an average of 3 microm before ceasing movement. Finally, we purified native **CENP -E**, a **kinesin**-related protein that has been implicated in chromosome congression during mitosis, and we found that this form of **CENP-E** does not induce microtubule gliding but is able to bind to microtubules.

L11 ANSWER 10 OF 30 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2001:165210 SCISEARCH
THE GENUINE ARTICLE: 400QK
TITLE: Chromosome movement in mitosis requires microtubule anchorage at spindle poles
AUTHOR: Gordon M B; Howard L; Compton D A (Reprint)
CORPORATE SOURCE: Dartmouth Med Sch, Dept Biochem, Hanover, NH 03755 USA (Reprint); Dartmouth Coll, Rippel Electron Microscope Facil, Hanover, NH 03755 USA
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CELL BIOLOGY, (5 FEB 2001) Vol. 152, No. 3, pp. 425-434.
Publisher: ROCKEFELLER UNIV PRESS, 1114 FIRST AVE, 4TH FL, NEW YORK, NY 10021 USA.
ISSN: 0021-9525.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 76

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Anchorage of microtubule minus ends at spindle poles has been proposed to bear the load of poleward forces exerted by kinetochore-associated **motors** so that chromosomes move toward the poles rather than the poles toward the chromosomes. To test this hypothesis, we monitored chromosome movement during mitosis after perturbation of nuclear mitotic apparatus protein (NuMA) and the **human** homologue of the KIN C **motor** family (HSET), two noncentrosomal proteins involved in spindle pole organization in animal cells. Perturbation of NuMA alone disrupts spindle pole organization and delays anaphase onset, but does not alter the velocity of oscillatory chromosome movement in prometaphase. Perturbation of HSET alone increases the duration of prometaphase, but does not alter the velocity of chromosome movement in prometaphase or anaphase. In contrast, simultaneous perturbation of both HSET and NuMA severely suppresses directed chromosome movement in prometaphase. Chromosomes coalesce near the center of these cells on bi-oriented spindles that lack organized poles. Immunofluorescence and electron microscopy verify microtubule attachment to sister kinetochores, but this attachment fails to generate proper tension across sister kinetochores.

These results demonstrate that anchorage of microtubule minus ends at spindle poles mediated by overlapping mechanisms involving both NuMA and HSET is essential for chromosome movement during mitosis.

L11 ANSWER 11 OF 30 MEDLINE on STN
ACCESSION NUMBER: 2001338615 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11250166
TITLE: Chromosome movement: dynein-out at the kinetochore.
AUTHOR: Banks J D; Heald R
CORPORATE SOURCE: Department of Molecular and Cell Biology, University of California, Berkeley, California 94720-3200, USA.. jenbanks@uclink4.berkeley.edu
SOURCE: Current biology : CB, (2001 Feb 20) 11 (4) R128-31. Ref: 28
Journal code: 9107782. ISSN: 0960-9822.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010618
Last Updated on STN: 20010618
Entered Medline: 20010614

AB Cell biologists have long speculated that a minus end-directed **motor** localized at kinetochores contributes to the poleward movement of chromosomes during mitosis. Two recent studies provide direct evidence that cytoplasmic dynein can perform this function.

L11 ANSWER 12 OF 30 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 1998167852 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9499420
TITLE: Localization of **motor**-related proteins and associated complexes to active, but not inactive, centromeres.
AUTHOR: Faulkner N E; Vig B; Echeverri C J; Wordeman L; Vallee R B
CORPORATE SOURCE: Cell Biology Group, Worcester Foundation for Biomedical Research, Shrewsbury, MA 01545, USA.
CONTRACT NUMBER: GM478434 (NIGMS)
SOURCE: Human molecular genetics, (1998 Apr) 7 (4) 671-7.
Journal code: 9208958. ISSN: 0964-6906.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980520
Last Updated on STN: 19980520
Entered Medline: 19980512

AB Multicentric chromosomes are often found in tumor cells and certain cell lines. How they are generated is not fully understood, though their stability suggests that they are non-functional during chromosome segregation. Growing evidence has implicated microtubule **motor** proteins in attachment of chromosomes to the mitotic spindle and in chromosome movement. To better understand the molecular basis for the inactivity of centromeres associated with secondary constrictions, we have tested these structures by immunofluorescence microscopy for the presence of **motor** complexes and associated proteins. We find strong immunoreactivity at the active, but not inactive, centromeres of prometaphase multicentric chromosomes using antibodies to the cytoplasmic dynein intermediate chains, three components of the dynactin complex (dynamitin, Arp1 and p150 Glued), the **kinesin**-related proteins **CENP-E** and MCAK and the proposed structural and

checkpoint proteins HZW10, CENP-F and Mad2p. These results offer new insight into the assembly and composition of both primary and secondary constrictions and provide a molecular basis for the apparent inactivity of the latter during chromosome segregation.

L11 ANSWER 13 OF 30 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1998:437989 SCISEARCH
THE GENUINE ARTICLE: ZR489
TITLE: Rigor-type mutation in the **kinesin**-related protein HsEg5 changes its subcellular localization and induces microtubule bundling
AUTHOR: Blangy A (Reprint); Chaussepied P; Nigg E A
CORPORATE SOURCE: CNRS, CRBM, IFR 24, 1919 ROUTE MENDE, F-34033 MONTPELLIER, FRANCE (Reprint); SWISS INST EXPT CANC RES, CH-1066 EPALINGES, SWITZERLAND; UNIV GENEVA, DEPT MOL BIOL, CH-1211 GENEVA, SWITZERLAND
COUNTRY OF AUTHOR: FRANCE; SWITZERLAND
SOURCE: CELL MOTILITY AND THE CYTOSKELETON, (FEB 1998) Vol. 40, No. 2, pp. 174-182.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0886-1544.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 54
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB HsEg5 is a **human kinesin**-related **motor** protein essential for the formation of a bipolar mitotic spindle. It interacts with the mitotic centrosomes in a phosphorylation-dependent manner. To investigate further the mechanisms involved in targetting HsEg5 to the spindle apparatus, we expressed various mutants of HsEg5 in HeLa cells. All these mutants share a mutation of Thr-112 in the N-terminal **motor** domain, resulting in the inactivation of the ATP binding domain. In vitro, the HsEg5-T112N mutant **motor** domain showed a nucleotide-independent microtubule association, typical of a **kinesin** protein binding to microtubules in a rigor state. In vivo, overexpression of the HsEg5 rigor mutant in HeLa cells induced, in interphase, microtubule bundling, and, in mitosis, the formation of monopolar mitotic spindles similar to those observed after microinjection of anti-HsEg5 antibodies. Localization of the HsEg5 rigor mutant on cytoplasmic microtubules did not require the C-terminal tail domain but was lost when the stalk domain was also deleted. Sucrose gradient centrifugation experiments showed that microtubule bundling was most likely caused by the binding of HsEg5 mutants in a dimeric state. These results demonstrate that the precise subcellular localization of HsEg5 in vivo is regulated not only by the phosphorylation of the tail domain but also by the oligomeric state of the protein. (C) 1998 Wiley-Liss, Inc.

L11 ANSWER 14 OF 30 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 97258096 EMBASE
DOCUMENT NUMBER: 1997258096
TITLE: **CENP-E** is an essential kinetochore **motor** in maturing oocytes and is masked during mos-dependent, cell cycle arrest at metaphase II.
AUTHOR: Duesbery N.S.; Choi T.; Brown K.D.; Wood K.W.; Resau J.; Fukasawa K.; Cleveland D.W.; Vande Woude G.F.
CORPORATE SOURCE: G.F. Vande Woude, ABL-Basic Research Program, National Cancer Institute, Frederick Cancer Res./Devt. Center, P.O. Box B, Frederick, MD 21702-1201, United States
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997) 94/17 (9165-9170).

Refs: 53
ISSN: 0027-8424 CODEN: PNASA6
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 021 Developmental Biology and Teratology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **CENP-E**, a **kinesin**-like protein that is known to associate with kinetochores during all phases of mitotic chromosome movement, is shown here to be a component of meiotic kinetochores as well. **CENP-E** is detected at kinetochores during metaphase I in both mice and frogs, and, as in mitosis, is relocalized to the midbody during telophase. **CENP-E** function is essential for meiosis I because injection of an antibody to **CENP-E** into mouse oocytes in prophase completely prevented progression of those oocytes past metaphase I. Beyond this, **CENP-E** is modified or masked during the natural, Mos- dependent, cell cycle arrest that occurs at metaphase II, although it is readily detectable at the kinetochores in metaphase II oocytes derived from mos-deficient (MOS(-/-)) mice that fail to arrest at metaphase II. This must reflect a masking of some **CENP-E** epitopes, not the absence of **CENP-E**, in meiosis II because a different polyclonal antibody raised to the tail of **CENP-E** detects **CENP-E** at kinetochores of metaphase II-arrested eggs and because **CENP-E** reappears in telophase of mouse oocytes activated in the absence of protein synthesis.

L11 ANSWER 15 OF 30 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 97361828 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9218789
TITLE: Identification of a **motor** protein required for filamentous growth in Ustilago maydis.
AUTHOR: Lehmler C; Steinberg G; Snetselaar K M; Schliwa M; Kahmann R; Bolker M
CORPORATE SOURCE: Institut fur Genetik und Mikrobiologie der Universitat Munchen, Germany.
SOURCE: EMBO journal, (1997 Jun) 16 (12) 3464-73.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L47106; GENBANK-U92844; GENBANK-U92845; SWISSPROT-Q02224
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970825
Last Updated on STN: 20030228
Entered Medline: 19970808
AB The phytopathogenic fungus Ustilago maydis exists in two stages, the yeast-like haploid form and the filamentous dikaryon. Both pathogenicity and dimorphism are genetically controlled by two mating-type loci, with only the filamentous stage being pathogenic on corn. We have identified two genes (kin1 and kin2) encoding **motor** proteins of the **kinesin** family. Kin1 is most similar to the **human** **CENP-E** gene product, while Kin2 is most closely related to the conventional **kinesin** Nkin of Neurospora crassa. Deletion mutants of kin1 had no discernible phenotype; delta kin2 mutants, however, were severely affected in hyphal extension and pathogenicity. The wild-type dikaryon showed rapid tip growth, with all the cytoplasm being moved to the tip compartment. Left behind are septate cell wall tubes devoid of cytoplasm. In delta kin2 mutants, dikaryotic cells were formed after cell fusion, but these hyphal structures remained short and filled with cytoplasm. A functional green fluorescent protein (GFP)-Kin2 fusion

was generated and used to determine the localization of the **motor** protein by fluorescence microscopy. Inspection of the hyphal tips by electron microscopy revealed a characteristic accumulation of darkly stained vesicles which was absent in mutant cells. We suggest that the **motor** protein Kin2 is involved in organizing this specialized growth zone at the hyphal tip, probably by affecting the vectorial transport of vesicles.

L11 ANSWER 16 OF 30 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 1998060834 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9396744
TITLE: **CENP-E** function at kinetochores is essential for chromosome alignment.
AUTHOR: Schaar B T; Chan G K; Maddox P; Salmon E D; Yen T J
CORPORATE SOURCE: Cell and Molecular Biology Graduate Group, University of Pennsylvania, Philadelphia, Pennsylvania 19103, USA.
CONTRACT NUMBER: CA06927 (NCI)
GM24364 (NIGMS)
GM44762 (NIGMS)
SOURCE: Journal of cell biology, (1997 Dec 15) 139 (6) 1373-82.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980129
Last Updated on STN: 19980129
Entered Medline: 19980113

AB **CENP-E** is a **kinesin**-like protein that binds to kinetochores and may provide functions that are critical for normal chromosome motility during mitosis. To directly test the in vivo function of **CENP-E**, we microinjected affinity-purified antibodies to block the assembly of **CENP-E** onto kinetochores and then examined the behavior of these chromosomes. Chromosomes lacking **CENP-E** at their kinetochores consistently exhibited two types of defects that blocked their alignment at the spindle equator. Chromosomes positioned near a pole remained mono-oriented as they were unable to establish bipolar microtubule connections with the opposite pole. Chromosomes within the spindle established bipolar connections that supported oscillations and normal velocities of kinetochore movement between the poles, but these bipolar connections were defective because they failed to align the chromosomes into a metaphase plate. Overexpression of a mutant that lacked the amino-terminal 803 amino acids of **CENP-E** was found to saturate limiting binding sites on kinetochores and competitively blocked endogenous **CENP-E** from assembling onto kinetochores. Chromosomes saturated with the truncated **CENP-E** mutant were never found to be aligned but accumulated at the poles or were strewn within the spindle as was the case when cells were microinjected with **CENP-E** antibodies. As the **motor** domain was contained within the portion of **CENP-E** that was deleted, the chromosomal defect is likely attributed to the loss of **motor** function. The combined data show that **CENP-E** provides kinetochore functions that are essential for monopolar chromosomes to establish bipolar connections and for chromosomes with connections to both spindle poles to align at the spindle equator. Both of these events rely on activities that are provided by **CENP-E**'s **motor** domain.

L11 ANSWER 17 OF 30 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1998:25462 SCISEARCH
THE GENUINE ARTICLE: YM669

TITLE: Localization of **CENP-E** in the fibrous corona and outer plate of mammalian kinetochores from prometaphase through anaphase
AUTHOR: Cooke C A; Schaar B; Yen T J; Earnshaw W C (Reprint)
CORPORATE SOURCE: UNIV EDINBURGH, INST CELL & MOL BIOL, MICHAEL SWANN BLDG, KINGS BLDG, MAYFIELD RD, EDINBURGH EH9 3JR, MIDLOTHIAN, SCOTLAND (Reprint); UNIV EDINBURGH, INST CELL & MOL BIOL, EDINBURGH EH9 3JR, MIDLOTHIAN, SCOTLAND; FOX CHASE CANC CTR, PHILADELPHIA, PA 19111
COUNTRY OF AUTHOR: SCOTLAND; USA
SOURCE: CHROMOSOMA, (1 DEC 1997) Vol. 106, No. 7, pp. 446-455. Publisher: SPRINGER VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010. ISSN: 0009-5915.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 22

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We have conducted a detailed ultrastructural analysis of the distribution of the **kinesin**-related centromere protein **CENP-E** during mitosis in cultured **human**, rat kangaroo and Indian muntjac cells. Using an affinity-purified polyclonal antibody and detection by 0.8 nm colloidal gold particles, **CENP-E** was localized primarily to the fibrous corona of the kinetochore in prometaphase and metaphase cells. Some labeling of the kinetochore outer plate was also observed. The distribution of fibrous corona-associated **CENP-E** did not change dramatically following the attachment of microtubules to the kinetochore. Thus, the normal disappearance of this kinetochore substructure in conventional electron micrographs of mitotic chromosomes with attached kinetochores is not due to the corona becoming stretched along the spindle microtubules as has been suggested. Examination of cells undergoing anaphase chromatid movement revealed the presence of **CENP-E** still associated with the outer surface of the kinetochore plate. At the same time, the majority of detectable **CENP-E** in these cells was associated with the bundles of antiparallel microtubules in the central spindle. **CENP-E** in this region of the cell is apparently associated with the stem body matrix material. The simultaneous localization of **CENP-E** on centromeres and the central spindle during anaphase was confirmed by both wide-field microscopy of **human** cells and conventional fluorescence microscopy of rat kangaroo cells. Together, the observations reported here are consistent with models in which **CENP-E** has a role in promoting the poleward migration of sister chromatids during anaphase A.

L11 ANSWER 18 OF 30 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 97477390 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9334346
TITLE: The microtubule-dependent **motor** centromere-associated protein E (**CENP-E**) is an integral component of kinetochore corona fibers that link centromeres to spindle microtubules.
AUTHOR: Yao X; Anderson K L; Cleveland D W
CORPORATE SOURCE: Laboratory of Cell Biology, Ludwig Institute for Cancer Research, School of Medicine, University of California, La Jolla, CA 92093-0660, USA.
CONTRACT NUMBER: GM 29513 (NIGMS)
SOURCE: Journal of cell biology, (1997 Oct 20) 139 (2) 435-47. Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971120

AB Centromere-associated protein E (**CENP-E**) is a **kinesin**-related microtubule **motor** protein that is essential for chromosome congression during mitosis. Using immunoelectron microscopy, **CENP-E** is shown to be an integral component of the kinetochore corona fibers that tether centromeres to the spindle. Immediately upon nuclear envelope fragmentation, an associated plus end **motor** trafficks cytoplasmic **CENP-E** toward chromosomes along astral microtubules that enter the nuclear volume. Before or concurrently with initial lateral attachment of spindle microtubules, **CENP-E** targets to the outermost region of the developing kinetochores. After stable attachment, throughout chromosome congression, at metaphase, and throughout anaphase A, **CENP-E** is a constituent of the corona fibers, extending at least 50 nm away from the kinetochore outer plate and intertwining with spindle microtubules. In congressing chromosomes, **CENP-E** is preferentially associated with (or accessible at) the stretched, leading kinetochore known to provide the primary power for chromosome movement. Taken together, this evidence strongly supports a model in which **CENP-E** functions in congression to tether kinetochores to the disassembling microtubule plus ends.

L11 ANSWER 19 OF 30 MEDLINE on STN
ACCESSION NUMBER: 1998028574 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9363944
TITLE: **CENP-E** is a plus end-directed kinetochore **motor** required for metaphase chromosome alignment.
AUTHOR: Wood K W; Sakowicz R; Goldstein L S; Cleveland D W
CORPORATE SOURCE: Laboratory of Cell Biology, Ludwig Institute for Cancer Research, University of California at San Diego, La Jolla 92093-0660, USA.
SOURCE: Cell, (1997 Oct 31) 91 (3) 357-66.
Journal code: 0413066. ISSN: 0092-8674.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF027728
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19980109
Entered Medline: 19971210

AB Mitosis requires dynamic attachment of chromosomes to spindle microtubules. This interaction is mediated largely by kinetochores. During prometaphase, forces exerted at kinetochores, in combination with polar ejection forces, drive congression of chromosomes to the metaphase plate. A major question has been whether kinetochore-associated microtubule **motors** play an important role in congression. Using immunodepletion from and antibody addition to Xenopus egg extracts, we show that the kinetochore-associated **kinesin**-like **motor** protein **CENP-E** is essential for positioning chromosomes at the metaphase plate. We further demonstrate that **CENP-E** powers movement toward microtubule plus ends in vitro. These findings support a model in which **CENP-E** functions in congression to tether kinetochores to dynamic microtubule plus ends.

L11 ANSWER 20 OF 30 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 97:370707 SCISEARCH

THE GENUINE ARTICLE: WX609
TITLE: Increased chromokinesin immunoreactivity in retinoblastoma cells
AUTHOR: Yan R T; Wang S Z (Reprint)
CORPORATE SOURCE: UNIV ALABAMA, SCH MED, EYE FDN HOSP, DEPT OPHTHALMOL, 700 S 18TH ST, BIRMINGHAM, AL 35233 (Reprint); UNIV ALABAMA, SCH MED, EYE FDN HOSP, DEPT OPHTHALMOL, BIRMINGHAM, AL 35233
COUNTRY OF AUTHOR: USA
SOURCE: GENE, (21 APR 1997) Vol. 189, No. 2, pp. 263-267. Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS. ISSN: 0378-1119.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 17

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB chromokinesin is a developmentally down-regulated gene with specific expression in proliferating cells during embryonic chick development. It encodes a DNA-binding **motor** protein localized along the chromosome arm during mitosis, suggesting that the protein may be a component of the long-observed, yet poorly understood 'ejection force' hypothesized to be involved in controlling the direction and speed of chromosome movement. We have isolated **human** chromokinesin; with affinity-purified antibodies we demonstrated immunocytochemically that Chromokinesin was present at a much higher level in cultured retinoblastoma cells than in primary cultures of **human** dermal fibroblasts. The increase in immunoreactivity was particularly prominent in interphase cells, whereas in primary cultures of fibroblasts immunopositive cells were predominantly M-phase cells. These observations imply a deregulation of chromokinesin in retinoblastoma cells. Data presented here may be useful in designing strategies to modulate chromosome movement and cell proliferation with either antisense oligonucleotides or specific antibodies, and hence may set the stage for further investigations of the involvement of chromosome **motor** molecules in mitosis under normal and pathological conditions. (C) 1997 Elsevier Science B.V.

L11 ANSWER 21 OF 30 MEDLINE on STN DUPLICATE 11
ACCESSION NUMBER: 96338605 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8743943
TITLE: The **kinesin**-like protein **CENP-E** is kinetochore-associated throughout poleward chromosome segregation during anaphase-A.
AUTHOR: Brown K D; Wood K W; Cleveland D W
CORPORATE SOURCE: Department of Biological Chemistry, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA.
CONTRACT NUMBER: GM 29513 (NIGMS)
SOURCE: Journal of cell science, (1996 May) 109 (Pt 5) 961-9. Journal code: 0052457. ISSN: 0021-9533.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961231

AB The **kinesin**-like protein **CENP-E** transiently associates with kinetochores following nuclear envelope breakdown in late prophase, remains bound throughout metaphase, but sometime after anaphase onset it releases and by telophase becomes bound to interzonal microtubules of the mitotic spindle. Inhibition of poleward chromosome

movement in vitro by **CENP-E** antibodies and association of **CENP-E** with minus-end directed microtubule motility in vitro have combined to suggest a key role for **CENP-E** as an anaphase chromosome **motor**. For this to be plausible in vivo depends on whether **CENP-E** remains kinetochore associated during anaphase. Using Indian muntjac cells whose seven chromosomes have large, easily tracked kinetochores, we now show that **CENP-E** is kinetochore-associated throughout the entirety of anaphase-A (poleward chromosome movement), relocating gradually during spindle elongation (anaphase-B) to the interzonal microtubules. These observations support roles for **CENP-E** not only in the initial alignment of chromosomes at metaphase and in spindle elongation in anaphase-B, but also in poleward chromosome movement in anaphase-A.

L11 ANSWER 22 OF 30 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 1996:524605 BIOSIS
DOCUMENT NUMBER: PREV199699246961
TITLE: Modulation of **CENP-E** organization at kinetochores by spindle microtubule attachment.
AUTHOR(S): Thrower, Douglas A. [Reprint author]; Jordan, Mary Ann; Wilson, Leslie
CORPORATE SOURCE: Dep. Mol., Cell. Dev. Biol., Univ. Calif., Santa Barbara, CA 93106, USA
SOURCE: Cell Motility and the Cytoskeleton, (1996) Vol. 35, No. 2, pp. 121-133.
CODEN: CMCYEO. ISSN: 0886-1544.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Nov 1996
Last Updated on STN: 22 Nov 1996

AB **CENP-E** is a protein of the **kinesin** superfamily that appears as small paired globules at kinetochores of chromosomes in mammalial cells during prometaphase and metaphase of mitosis (Yen et al., 1992: Nature 359:536-539). In the present study we found that a significant number of chromosomes during early prometaphase in HeLa cells (approximately 30%) were stained with a **CENP-E** antibody in the form of large C-shaped "collars" that partially encircled the chromosomes. The C-shaped **CENP-E** collars were present only transiently and were completely replaced by small paired globular forms prior to metaphase. Most chromosomes had persistent **CENP-E** collars in cells blocked at mitosis with a vinblastine concentration sufficient to prevent all microtubule formation. Attachment of newly formed microtubules to the kinetochores after removal of vinblastine resulted in loss of the collars and replacement with small paired globules. Similarly, a higher proportion of chromosomes isolated from vinblastine-treated cells contained **CENP-E** collars (73%), and the "capture" (i.e., attachment) of microtubules by the chromosomes resulted in conversion of the collars into small paired globules in vitro. Thus, the **CENP-E** collars form prior to microtubule attachment and disappear after attachment of the chromosomes to the spindle. The **CENP-E** collars may facilitate capture of microtubules by chromosomes during prometaphase.

L11 ANSWER 23 OF 30 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 95:329093 SCISEARCH
THE GENUINE ARTICLE: QY118
TITLE: CHARACTERIZATION OF A MINUS END-DIRECTED **KINESIN**-LIKE **MOTOR** PROTEIN FROM CULTURED-MAMMALIAN-CELLS
AUTHOR: KURIYAMA R (Reprint); KOFRON M; ESSNER R; KATO T; DRAGASGRANOIC S; OMOTO C K; KHODJAKOV A

CORPORATE SOURCE: UNIV MINNESOTA, DEPT CELL BIOL & NEUROANAT, 4-135 JACKSON HALL, 321 CHURCH ST SE, MINNEAPOLIS, MN, 55455 (Reprint); WASHINGTON STATE UNIV, DEPT GENET & CELL BIOL, PULLMAN, WA, 99164
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CELL BIOLOGY, (MAY 1995) Vol. 129, No. 4, pp. 1049-1059.
ISSN: 0021-9525.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 51

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Using the CHO2 monoclonal antibody raised against CHO spindles (Sellitto, C., M. Kimble, and R. Kuriyama. 1992. Cell Motil. Cytoskeleton. 22:7-24) we identified a 66-kD protein located at the interphase centrosome and mitotic spindle. Isolated cDNAs for the antigen encode a 622-amino acid polypeptide. Sequence analysis revealed the presence of 340-amino acid residues in the COOH terminus, which is homologous to the **motor** domain conserved among other members of the **kinesin** superfamily. The protein is composed of a central alpha-helical portion with globular domains at both NH2 and COOH termini, and the epitope to the monoclonal antibody resides in the central alpha-helical stalk. A series of deletion constructs were created for in vitro analysis of microtubule interactions. While the microtubule binding and bundling activities require both the presence of the COOH terminus and the alpha-helical domain, the NH2-terminal half of the antigen lacked the ability to interact with microtubules. The full-length as well as deleted proteins consisting of the COOH-terminal **motor** and the central alpha-helical stalk supported microtubule gliding, with velocity ranging from 1.0 to 8.4 mu m/minute. The speed of microtubule movement decreased with decreasing lengths of the central stalk attached to the COOH-terminal **motor**. The microtubules moved with their plus end leading, indicating that the antigen is a minus end-directed **motor**. The CHO2 sequence shows 86% identify to HSET, a gene located at the centromeric end of the **human** MHC region in chromosome 6 (Ando, A., Y. Y. Kikuti, H. Kawata, N. Okamoto, T. Imai, T. Eki, K. Yokoyama, E. Soeda, T. Ikemura, K. Abe, and H. Inoko. 1994. Immunogenetics. 39:194-200), indicating that HSET might represent a **human** homologue of the CHO2 antigen.

L11 ANSWER 24 OF 30 MEDLINE on STN DUPLICATE 12
ACCESSION NUMBER: 95196755 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7889940
TITLE: Mitotic HeLa cells contain a **CENP-E** -associated minus end-directed microtubule **motor**.
AUTHOR: Thrower D A; Jordan M A; Schaar B T; Yen T J; Wilson L
CORPORATE SOURCE: Department of Biological Sciences, University of California, Santa Barbara 93106.
CONTRACT NUMBER: CA06927 (NCI)
GM44762 (NIGMS)
SOURCE: EMBO journal, (1995 Mar 1) 14 (5) 918-26.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 19950427
Last Updated on STN: 19970203
Entered Medline: 19950420

AB A minus end-directed microtubule **motor** activity from extracts of HeLa cells blocked at prometaphase/metaphase of mitosis with vinblastine has been partially purified and characterized. The **motor**

activity was eliminated by immunodepletion of Centromere binding protein E (**CENP-E**). The **CENP-E**-associated **motor** activity, which was not detectable in interphase cells, moved microtubules at mean rates of 0.46 micron/s at 37 degrees C and 0.24 micron/s at 25 degrees C. The **motor** activity co-purified with **CENP-E** through several purification procedures. **Motor** activity was clearly not due to dynein or to **kinesin**. The microtubule gliding rates of the **CENP-E**-associated **motor** were different from those of dynein and **kinesin**. In addition, the pattern of nucleotide substrate utilization by the **CENP-E**-associated **motor** and the sensitivity to inhibitors were different from those of dynein and **kinesin**. The **CENP-E**-associated **motor** had an apparent native molecular weight of 874,000 Da and estimated dimensions of 2 nm x 80 nm. This is the first demonstration of **motor** activity associated with **CENP-E**, strongly supporting the hypothesis that **CENP-E** may act as a minus end-directed microtubule **motor** during mitosis.

L11 ANSWER 25 OF 30 MEDLINE on STN
ACCESSION NUMBER: 95122643 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7822426
TITLE: Identification and partial characterization of mitotic centromere-associated **kinesin**, a **kinesin**-related protein that associates with centromeres during mitosis.
COMMENT: Comment in: J Cell Biol. 1995 Jan;128(1-2):1-4. PubMed ID: 7822407
AUTHOR: Wordeman L; Mitchison T J
CORPORATE SOURCE: Department of Physiology and Biophysics, University of Washington, Seattle 98195.
CONTRACT NUMBER: CA-09270 (NCI)
GM-39565 (NIGMS)
SOURCE: Journal of cell biology, (1995 Jan) 128 (1-2) 95-104.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950223
Last Updated on STN: 20021227
Entered Medline: 19950214

AB Using antipeptide antibodies to conserved regions of the **kinesin** **motor** domain, we cloned a **kinesin**-related protein that associates with the centromere region of mitotic chromosomes. We call the protein MCAK, for mitotic centromere-associated **kinesin**. MCAK appears concentrated on centromeres at prophase and persists until telophase, after which time the localization disperses. It is found throughout the centromere region and between the kinetochore plates of isolated mitotic CHO chromosomes, in contrast to two other kinetochore-associated microtubule **motors**: cytoplasmic dynein and **CENP-E** (Yen et al., 1992), which are closer to the outer surface of the kinetochore plates. Sequence analysis shows MCAK to be a **kinesin**-related protein with the **motor** domain located in the center of the protein. It is 60-70% similar to kif2, a **kinesin**-related protein originally cloned from mouse brain with a centrally located **motor** domain (Aizawa et al., 1992). MCAK protein is present in interphase and mitotic CHO cells and is transcribed as a single 3.4-kb message.

L11 ANSWER 26 OF 30 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 95:65950 SCISEARCH

THE GENUINE ARTICLE: QB175
TITLE: HETEROGENEITY AND MICROTUBULE INTERACTION OF THE CHO1 ANTIGEN, A MITOSIS-SPECIFIC **KINESIN**-LIKE PROTEIN - ANALYSIS OF SUBDOMAINS EXPRESSED IN INSECT SF9 CELLS
AUTHOR: KURIYAMA R (Reprint); DRAGASGRANOIC S; MAEKAWA T; VASSILEV A; KHODJAKOV A; KOBAYASHI H
CORPORATE SOURCE: UNIV MINNESOTA, DEPT CELL BIOL & NEUROANAT, 4-135 JACKSON HALL, 321 CHURCH ST SE, MINNEAPOLIS, MN, 55455 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CELL SCIENCE, (DEC 1994) Vol. 107, Part 12, pp. 3485-3499.
ISSN: 0021-9533.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 52

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The CHO1 antigen is a mitosis-specific **kinesin**-like **motor** located at the interzonal region of the spindle. The **human** cDNA coding for the antigen contains a domain with sequence similarity to the **motor** domain of **kinesin**-like protein (Nislow et al., Nature 359, 543, 1992). Here we cloned cDNAs encoding the CHO1 antigen by immunoscreening of a CHO Uni-Zap expression library, the same species in which the original monoclonal antibody was raised, cDNAs of CHO cells encode a 953 amino acid polypeptide with a calculated molecular mass of 109 kDa. The N-terminal 73% of the antigen was 87% identical to the **human** clone, whereas the remaining 27% of the coding region showed only 48% homology. Insect Sf9 cells infected with baculovirus containing the full-length insert produced 105 and 95 kDa polypeptides, the same doublet identified as the original antigen in CHO cells. Truncated polypeptides corresponding to the N-terminal **motor** and C-terminal tail produced a 56 and 54 kDa polypeptide in Sf9 cells, respectively. Full and N-terminal proteins co-sedimented with, and caused bundling of, brain microtubules in vitro, whereas the C-terminal polypeptide did not. Cells expressing the N terminus formed one or more cytoplasmic processes. Immunofluorescence as well as electron microscopic observations revealed the presence of thick bundles of microtubules, which were closely packed, forming a marginal ring just beneath the cell membrane and a core in the processes. The diffusion coefficient and sedimentation coefficient were determined for the native CHO1 antigen by gel filtration and sucrose density gradient centrifugation, respectively. The native molecular mass of overinduced protein in Sf9 cells was calculated as 219 kDa, suggesting that the antigen exists as a dimer. Intrinsic CHO1 antigen in cultured mammalian cells forms a larger native complex (native molecular mass, 362 kDa), which may suggest the presence of additional molecule(s) associating with the CHO1 **motor** molecule.

L11 ANSWER 27 OF 30 MEDLINE on STN DUPLICATE 13
ACCESSION NUMBER: 94266962 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8207059
TITLE: Cyclin-like accumulation and loss of the putative kinetochore **motor CENP-E** results from coupling continuous synthesis with specific degradation at the end of mitosis.
AUTHOR: Brown K D; Coulson R M; Yen T J; Cleveland D W
CORPORATE SOURCE: Department of Biological Chemistry, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205.
CONTRACT NUMBER: GM 29513 (NIGMS)
SOURCE: Journal of cell biology, (1994 Jun) 125 (6) 1303-12.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199407
ENTRY DATE: Entered STN: 19940721
Last Updated on STN: 19970203
Entered Medline: 19940712

AB **CENP-E** is a **kinesin**-like protein that binds to kinetochores through the early stages of mitosis, but after initiation of anaphase, it relocalizes to the overlapping microtubules in the midzone, ultimately concentration in the developing midbody. By immunoblotting of cells separated at various positions in the cell cycle using centrifugal elutriation, we show that **CENP-E** levels increase progressively across the cycle peaking at approximately 22,000 molecules/cell early in mitosis, followed by an abrupt (> 10 fold) loss at the end of mitosis. Pulse-labeling with [35S]methionine reveals that beyond a twofold increase in synthesis between G1 and G2, interphase accumulation results primarily from stabilization of **CENP-E** during S and G2. Despite localizing in the midbody during normal cell division, **CENP-E** loss at the end of mitosis is independent of cytokinesis, since complete blockage of division with cytochalasin has no affect on **CENP-E** loss at the M/G1 transition. Thus, like mitotic cyclins, **CENP-E** accumulation peaks before cell division, and it is specifically degraded at the end of mitosis. However, **CENP-E** degradation kinetically follows proteolysis of cyclin B in anaphase. Combined with cyclin A destruction before the end of metaphase, degradation of as yet unidentified components at the metaphase/anaphase transition, and cyclin B degradation at or after the anaphase transition, **CENP-E** destruction defines a fourth point in a mitotic cascade of timed proteolysis.

L11 ANSWER 28 OF 30 MEDLINE on STN
ACCESSION NUMBER: 94294810 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8023161
TITLE: Mitotic regulation of microtubule cross-linking activity of **CENP-E** kinetochore protein.
AUTHOR: Liao H; Li G; Yen T J
CORPORATE SOURCE: Fox Chase Cancer Center, Philadelphia, PA 19111.
CONTRACT NUMBER: CA-06927 (NCI)
GM-44762-02 (NIGMS)
SOURCE: Science, (1994 Jul 15) 265 (5170) 394-8.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 19940815
Last Updated on STN: 19940815
Entered Medline: 19940802

AB **CENP-E** is a **kinesin**-like protein that is transiently bound to kinetochores during early mitosis, becomes redistributed to the spindle midzone at anaphase, and is degraded after cytokinesis. At anaphase, **CENP-E** may cross-link the interdigitating microtubules in the spindle midzone through a **motor**-like binding site at the amino terminus and a 99-amino acid carboxyl-terminal domain that bound microtubules in a distinct manner. Phosphorylation of the carboxyl terminus by the mitotic kinase maturation promoting factor (MPF) inhibited microtubule-binding activity before anaphase. Thus, MPF suppresses the microtubule cross-linking activity of **CENP-E** until anaphase, when its activity is lost.

L11 ANSWER 29 OF 30 MEDLINE on STN
ACCESSION NUMBER: 94168458 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8122906

TITLE: With apologies to scheherazade: tails of 1001 **kinesin motors**.
AUTHOR: Goldstein L S
CORPORATE SOURCE: Department of Cellular and Developmental Biology, Harvard University, Cambridge, Massachusetts 02138.
SOURCE: Annual review of genetics, (1993) 27 319-51. Ref: 98
Journal code: 0117605. ISSN: 0066-4197.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199404
ENTRY DATE: Entered STN: 19940412
Last Updated on STN: 19940412
Entered Medline: 19940404

L11 ANSWER 30 OF 30 MEDLINE on STN DUPLICATE 14
ACCESSION NUMBER: 93024922 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1406971
TITLE: **CENP-E** is a putative kinetochore **motor** that accumulates just before mitosis.
COMMENT: Comment in: Nature. 1992 Oct 8;359(6395):480-2. PubMed ID: 1406965
AUTHOR: Yen T J; Li G; Schaar B T; Szilak I; Cleveland D W
CORPORATE SOURCE: Fox Chase Cancer Center, Philadelphia, Pennsylvania 19111.
SOURCE: Nature, (1992 Oct 8) 359 (6395) 536-9.
Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199211
ENTRY DATE: Entered STN: 19930122
Last Updated on STN: 19930122
Entered Medline: 19921113

AB The mechanics of chromosome movement, mitotic spindle assembly and spindle elongation have long been central questions of cell biology. After attachment in prometaphase of a microtubule from one pole, duplicated chromosome pairs travel towards the pole in a rapid but discontinuous motion. This is followed by a slower congression towards the midplate as the chromosome pair orients with each kinetochore attached to the microtubules from the nearest pole. The pairs disjoin at anaphase and translocate to opposite poles and the interpolar distance increases. Here we identify **CENP-E** as a **kinesin**-like **motor** protein (M(r) 312,000) that accumulates in the G2 phase of the cell cycle. **CENP-E** associates with kinetochores during congression, relocates to the spindle midzone at anaphase, and is quantitatively discarded at the end of the cell division. **CENP-E** is likely to be one of the **motors** responsible for mammalian chromosome movement and/or spindle elongation.

=> e bearud c/au
E1 1 BEARTUSK K/AU
E2 1 BEARTUSK KAETZ/AU
E3 0 --> BEARUD C/AU
E4 1 BEARULIEU C/AU
E5 6 BEARUP A J/AU
E6 2 BEARUP R S/AU
E7 1 BEARWALD J P/AU
E8 1 BEARWELL C G/AU
E9 1 BEARWOOD C J/AU

```
E10           7      BEARWOOD D/AU
E11           3      BEARWOOD D E/AU
E12           1      BEARWOOD DANA/AU

=> e beraud c/au
E1            1      BERAUD ALEXANDRE/AU
E2            9      BERAUD B/AU
E3          478 --> BERAUD C/AU
E4            1      BERAUD C L/AU
E5            9      BERAUD CASSEL A M/AU
E6            3      BERAUD CATHERINE/AU
E7            1      BERAUD CEDRIC FRANCIS/AU
E8            1      BERAUD CH J/AU
E9          120      BERAUD CHRISTOPHE/AU
E10           4      BERAUD CL/AU
E11          19      BERAUD COLOMB E/AU
E12           1      BERAUD COLOMB ELIAINE/AU

=> s e3
L12         478 "BERAUD C"/AU

=> e ohashi c/au
E1            1      OHASHI B/AU
E2            1      OHASHI B H/AU
E3           26 --> OHASHI C/AU
E4            2      OHASHI C B/AU
E5           15      OHASHI C T/AU
E6            8      OHASHI CARA/AU
E7            7      OHASHI CARA T/AU
E8            1      OHASHI CHE/AU
E9            1      OHASHI CHIAKI/AU
E10           3      OHASHI CHIE/AU
E11           1      OHASHI CHIGEO/AU
E12           1      OHASHI CHIGIRU/AU

=> s e3
L13          26 "OHASHI C"/AU

=> e sakowicz r/au
E1            1      SAKOWICZ MAREK/AU
E2           18      SAKOWICZ MONIKA/AU
E3           71 --> SAKOWICZ R/AU
E4            2      SAKOWICZ ROBERT/AU
E5           76      SAKOWICZ ROMAN/AU
E6            2      SAKOWICZ S/AU
E7           30      SAKOWICZ T/AU
E8           19      SAKOWICZ TOMASZ/AU
E9            1      SAKOWITSC/AU
E10           1      SAKOWITSCH K/AU
E11           1      SAKOWITSCH M/AU
E12           1      SAKOWITSCH W/AU

=> s e5
L14          76 "SAKOWICZ ROMAN"/AU

=> e vaisberg e/au
E1            3      VAISBERG CHAIKA/AU
E2            1      VAISBERG D/AU
E3           30 --> VAISBERG E/AU
E4           49      VAISBERG E A/AU
E5            3      VAISBERG E F/AU
E6            5      VAISBERG E I/AU
E7           23      VAISBERG E S/AU
E8            9      VAISBERG E V/AU
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E9           17     VAISBERG ELENA/AU
E10           2     VAISBERG ELENA V/AU
E11           4     VAISBERG EUGENI/AU
E12          17     VAISBERG EUGENI A/AU

=> s e3
L15          30 "VAISBERG E"/AU

=> e wood k/au
E1            1     WOOD JUSTIN A/AU
E2            2     WOOD JUSTIN G/AU
E3          803 --> WOOD K/AU
E4          192     WOOD K A/AU
E5           86     WOOD K B/AU
E6           45     WOOD K C/AU
E7           20     WOOD K D/AU
E8           85     WOOD K E/AU
E9           23     WOOD K F/AU
E10          25     WOOD K G/AU
E11          20     WOOD K H/AU
E12           2     WOOD K H B/AU

=> s e3
L16         803 "WOOD K"/AU

=> e yu m/au
E1            1     YU LYNN K/AU
E2            1     YU LYU/AU
E3         2350 --> YU M/AU
E4            1     YU M */AU
E5           23     YU M A/AU
E6           86     YU M B/AU
E7          959     YU M C/AU
E8           12     YU M C Z/AU
E9            2     YU M CHRISTINA/AU
E10          30     YU M D/AU
E11           8     YU M E/AU
E12           1     YU M ELISA/AU

=> s e3
L17        2350 "YU M"/AU

=> d his

     (FILE 'HOME' ENTERED AT 08:59:27 ON 17 SEP 2004)

     FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,
     LIFESCI' ENTERED AT 08:59:47 ON 17 SEP 2004
L1         14477 S KINESIN?
L2           831 S "CENP-E"
L3             1 S "CENTROMER BINDING"
L4             0 S CENTROMER (2W) "PROTEIN E"
L5           282 S L1 AND L2
L6           125 S HUMAN AND L5
L7            67 S MOTOR AND L6
L8        333307 S ATPASE
L9             6 S L6 AND L8
L10            6 DUP REM L9 (0 DUPLICATES REMOVED)
L11           30 DUP REM L7 (37 DUPLICATES REMOVED)
                 E BEARUD C/AU
                 E BERAUD C/AU
L12          478 S E3
                 E OHASHI C/AU
L13           26 S E3
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                E SAKOWICZ R/AU
L14           76 S E5
                E VAISBERG E/AU
L15           30 S E3
                E WOOD K/AU
L16          803 S E3
                E YU M/AU
L17         2350 S E3

=> s l11 or l12 or l13 or l14 or l15 or l16 or l17
L18        3786 L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17

=> s l2 and l18
L19          36 L2 AND L18

=> dup rem l19
PROCESSING COMPLETED FOR L19
L20          34 DUP REM L19 (2 DUPLICATES REMOVED)

=> d 1-34 ibib ab

L20  ANSWER 1 OF 34  EMBASE  COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
     on STN
ACCESSION NUMBER:   2004325682  EMBASE
TITLE:              Gene silencing of CENP-E by small
                    interfering RNA in HeLa cells leads to missegregation of
                    chromosomes after a mitotic delay.
AUTHOR:             Tanudji M.; Shoemaker J.; L'Italien L.; Russell L.; Chin
                    G.; Schebye X.M.
CORPORATE SOURCE:   X.M. Schebye, DNAX Research Institute, Palo Alto, CA 94304,
                    United States. xiaomin.schebye@dnax.org
SOURCE:             Molecular Biology of the Cell, (2004) 15/8 (3771-3781).
                    Refs: 33
                    ISSN: 1059-1524  CODEN: MBCEEV
COUNTRY:            United States
DOCUMENT TYPE:      Journal; Article
FILE SEGMENT:       029     Clinical Biochemistry
LANGUAGE:           English
SUMMARY LANGUAGE:   English
AB   Centromeric protein-E (CENP-E) is a kinesin
     -like motor protein required for chromosome congression at
     prometaphase. Functional perturbation of CENP-E by
     various methods results in a consistent phenotype, i.e., unaligned
     chromosomes during mitosis. One unresolved question from previous studies
     is whether cells complete mitosis or sustain mitotic arrest in the
     presence of unaligned chromosomes. Using RNA interference and
     video-microscopy, we analyzed the dynamic process of mitotic progression
     of HeLa(H2B)-GFP cells lacking CENP-E. Our results
     demonstrate that these cells initiated anaphase after a delayed mitotic
     progression due to the presence of unaligned chromosomes. In some dividing
     cells, unaligned chromosomes are present during anaphase, causing
     nondisjunction of some sister chromatids producing aneuploid daughter
     cells. Unlike in Xenopus extract, the loss of CENP-E
     in HeLa cells does not impair gross checkpoint activation because cells
     were arrested in mitosis in response to microtubule-interfering agents.
     However, the lack of CENP-E at kinetochores reduced
     the hyperphosphorylation of BubR1 checkpoint protein during mitosis, which
     may explain the loss of sensitivity of a cell to a few unaligned
     chromosomes in the absence of CENP-E. We also found
     that presynchronization with nocodazole sensitizes cells to the depletion
     of CENP-E, leading to more unaligned chromosomes,
     longer arrest, and cell death.

L20  ANSWER 2 OF 34     MEDLINE on STN
```

ACCESSION NUMBER: 2004258559 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 15159587
TITLE: Crystallization and preliminary crystallographic analysis of the **motor** domain of **human** kinetochore-associated protein **CENP-E** using an automated crystallization procedure.
AUTHOR: Garcia-Saez Isabel; Blot Delphine; Kahn Richard; Kozielski Frank
CORPORATE SOURCE: Laboratoire de Microscopie Electronique Structurale, Institut de Biologie Structurale Jean-Pierre Ebel (CEA-CNRS-UJF), 41 Rue Jules Horowitz, 38027 Grenoble CEDEX 01, France.. isabel.garcia@ibs.fr
SOURCE: Acta crystallographica. Section D, Biological crystallography, (2004 Jun) 60 (Pt 6) 1158-60. Journal code: 9305878. ISSN: 0907-4449.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20040526
Last Updated on STN: 20040629

AB **Human** centromere-associated protein E, a member of the **kinesin** superfamily, is a microtubule-dependent **motor** protein involved in cell division that has been localized transiently to the kinetochore. The protein is thought to be responsible for the correct attachment and positioning of chromosomes to the mitotic spindle during the metaphase. The 312 kDa protein comprises four different domains. In this study, the focus was on the N-terminal **motor** domain, which includes the ATP-binding site and a region for microtubule binding. Crystals of the **CENP-E motor** domain have been obtained by high-throughput crystallization screening using an automated TECAN crystallization robot. The crystals (737 x 132 x 79 microm) belong to the space group P2(1), with unit-cell parameters a = 49.35, b = 83.70, c = 94.16 angstroms, beta = 103.05 degrees. They diffract to 2.1 angstroms resolution using synchrotron radiation.

L20 ANSWER 3 OF 34 MEDLINE on STN
ACCESSION NUMBER: 2004334945 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15236970
TITLE: Crystal structure of the **motor** domain of the **human** kinetochore protein **CENP-E**
AUTHOR: Garcia-Saez Isabel; Yen Tim; Wade Richard H; Kozielski Frank
CORPORATE SOURCE: Laboratoire de Microscopie Electronique Structurale, Institut de Biologie Structurale, 41 rue Jules Horowitz, 38027 Grenoble Cedex 01, France.
CONTRACT NUMBER: CA06927 (NCI)
CA75138 (NCI)
GM44762 (NIGMS)
SOURCE: Journal of molecular biology, (2004 Jul 23) 340 (5) 1107-16. Journal code: 2985088R. ISSN: 0022-2836.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200408
ENTRY DATE: Entered STN: 20040707
Last Updated on STN: 20040826
Entered Medline: 20040825

AB The **human** kinetochore is a highly complex macromolecular structure that connects chromosomes to spindle microtubules (MTs) in order

to facilitate accurate chromosome segregation. Centromere-associated protein E (**CENP-E**), a member of the **kinesin** superfamily, is an essential component of the kinetochore, since it is required to stabilize the attachment of chromosomes to spindle MTs, to develop tension across aligned chromosomes, to stabilize spindle poles and to satisfy the mitotic checkpoint. Here we report the 2.5A resolution crystal structure of the **motor** domain and linker region of **human CENP-E** with MgADP bound in the active site. This structure displays subtle but important differences compared to the structures of **human** Eg5 and conventional **kinesin** . Our structure reveals that the **CENP-E** linker region is in a "docked" position identical to that in the **human** plus-end directed conventional **kinesin**. **CENP-E** has many advantages as a potential anti-mitotic drug target and this crystal structure of **human CENP-E** will provide a starting point for high throughput virtual screening of potential inhibitors.

L20 ANSWER 4 OF 34 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2004:7637 BIOSIS
DOCUMENT NUMBER: PREV200400008401
TITLE: Plus end-directed microtubule motor required for chromosome congression.
AUTHOR(S): Wood, Kenneth W. [Inventor, Reprint Author]; **Sakowicz, Roman** [Inventor]; Goldstein, Lawrence S. B. [Inventor]; Cleveland, Don W. [Inventor]
CORPORATE SOURCE: Delmar, CA, USA
ASSIGNEE: The Regents of the University of California
PATENT INFORMATION: US 6645748 November 11, 2003
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Nov 11 2003) Vol. 1276, No. 2. http://www.uspto.gov/web/menu/patdata.html. e-file. ISSN: 0098-1133 (ISSN print).
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Dec 2003
Last Updated on STN: 17 Dec 2003
AB The invention provides isolated nucleic acid and amino acid sequences of Xenopus **CENP-E** (XCENP-E), antibodies to XCENP-E, methods of screening for **CENP-E** modulators using biologically active **CENP-E**, and kits for screening for **CENP-E** modulators.

L20 ANSWER 5 OF 34 BIOTECHDS COPYRIGHT 2004 THE THOMSON CORP/ISI on STN
ACCESSION NUMBER: 2003-15387 BIOTECHDS
TITLE: Treatment of disease e.g. cancer, rheumatoid arthritis, Alzheimer's disease and Parkinson's disease involves administration of antisense oligonucleotide; **human kinesin**-specific oligonucleotide transfer and expression in host cell for gene therapy
AUTHOR: REINHARD C; WALTER A
PATENT ASSIGNEE: CHIRON CORP
PATENT INFO: WO 2003030832 17 Apr 2003
APPLICATION INFO: WO 2002-US32596 11 Oct 2002
PRIORITY INFO: US 2001-328444 12 Oct 2001; US 2001-328444 12 Oct 2001
DOCUMENT TYPE: Patent
LANGUAGE: English
OTHER SOURCE: WPI: 2003-381676 [36]
AB DERWENT ABSTRACT:
NOVELTY - Treatment of disease involves administering an antisense oligonucleotide. The oligonucleotide inhibits the expression of **human kinesin** gene. The **human kinesin** gene is **CENP-E**, **human** Eg5 or MCAK.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following: (1) an anti-sense oligonucleotide (I) having nucleic acid sequence CCTCCGCCATCCTATCAGGCTGAA, CCGAGGAGAAAGCGAAATAGGGAAG, GAGACCGACTCTTGCTCTGTTGCC, GTTGATCTGGGCTCGCAGAGGTAAT, CTCTGTGGTGTCGTACCTGTTGGGA, TGGGTTCAAGTGATTCTCGTGCCTC, TGTCAGCCAATCCTCCAGTTCGTAC, TTGTACGCCCTCCAAGAGAATCCTG, GCTCAAGCAATCCACCCGCCTCAG, GGGATTACAGGCATGAGCCACCGC, CACTCCATTTTTCTCACGGGCTGCA, CATTCTCCTGAGCCGTGATGCGAA, ACGGAACGGGGTGTGAGCCTTGT, TGTCAGCTTGCTCTCACGGAACGG, GGAGCTTATGCCTGGTGAGATCGTG, GAGTCAGCAAGGAAGAGAAACGCG, TGGATAAATTGCCTGGAATCAGCGC and CGTTGGATCTTGATAGCGAGACCGG (2) combination therapy involving administration of at least one chemotherapeutic or radionuclide and further involves administration of at least one anti-sense oligonucleotide, the oligonucleotide is administered either separately or in combination; and (3) a pharmaceutical composition comprising (I) and a carrier.

ACTIVITY - Cytostatic; Immunosuppressive; Virucide; Vasotropic; Cerebroprotective; Cardiant; Antibacterial; Fungicide; Protozoacide; Antirheumatic; Antiarthritic; Antiinflammatory; Anticonvulsant; Antiparkinsonian; Nootropic; Neuroprotective; Neuroleptic; CNS-Gen.; Sedative; Dermatological; Analgesic; Tranquilizer; Antidiabetic; Antilipemic; Nephrotropic; Gastrointestinal-Gen.; Antiulcer; Anti-HIV; Antiallergic; Antianemic; Osteopathic; Anthelmintic; Ophthalmological; Antithyroid; Respiratory-Gen.

MECHANISM OF ACTION - **Human kinesin** gene inhibitor; Modulator of function of nucleic acid molecule encoding **human kinesin**; Anchorage independent growth inhibitor.
The antisense oligonucleotide of sequence TGGATAAATTGCCTGGAATCAGCGC (i) was transfected into **human** colon cancer cell line SW620. The same colon cancer cell line was transfected with the corresponding reverse control sequence CGCGACTAAGGTCCGTTAAATAGGT (ii). The total number of colonies normalized were: for (i) was approximately 425 and for (ii) was approximately 800. The results showed that the antisense oligonucleotide inhibited the capability of the cells to grow in soft agar and inhibited anchorage independent growth. The results showed that the **kinesin** antisense oligonucleotide inhibited tumorigenesis.

USE - For treatment of disease having aberrant cell proliferation such as cancer e.g. colon cancer, T and B cell lymphoma, pancreatic cancer, breast cancer, leukemia, bladder cancer, stomach cancer, brain cancer, esophageal cancer, liver cancer, adrenalcarcinoma, lung cancer, testicular cancer, heart cancer, ovarian cancer, uterine cancer, head and neck cancer, bone cancer, cervical cancer, gall bladder cancer, parathrnoid cancer, penile cancer, prostate cancer, skin cancer, spleen cancer, thymus cancer, thyroid cancer, muscle cancer, ganglial cancer, melanoma, myeloma sarcoma and teratocarcinomas, digestive cancer, lymphoma, autoimmune disorder, viral infection, neurological disorder, condition associated with ischemia and liver or pancreatic disease (claimed), myocardial infarction and stroke. The neurological disorders e.g. epilepsy, ischemic cerebrovascular disease, cerebral neoplasm, Alzheimer's disease, Pick's disease, Huntington's disease, dementia, Parkinson's disease, extrapyramidal disorder, amyotrophic lateral sclerosis, **motor** neuron disorders, progressive neural muscular atrophy, retinitis pigmentosa, hereditary ataxia, suppurative intracranial thrombophlebitis, multiple sclerosis, demyelinating disease, bacterial and viral meningitis, brain abscess, subdural empyema, myelitis, paralysis, viral central nervous system disease, prion disease including kuru, Creutzfeldt-Jakob disease, Gerstmann-Straussler-Scheinker syndrome, insomnia, neurofibromatosis, mental retardation, cerebral palsy, autonomic nervous system disorder, muscular dystrophy, peripheral nervous system disorders, dermatomyositis, anxiety, schizophrenia, amnesia, diabetic neuropathy, tardive dyskinesia, Tourette's disease, cystic fibrosis, hypercholesterolemia, diabetic mellitus, hyper- and hypoglycemia, Grave's disease, neuralgia, Cushing's disease, Addison's disease, gastrointestinal disorders e.g. ulcerative colitis, duodenal

ulcer, AIDS, allergic reactions, autoimmune hemolytic anemia, proliferative glomerulonephritis, inflammatory bowel disease, myasthenia gravis, rheumatoid arthritis, osteoarthritis, scleroderma, Sjogren's syndrome, systemic lupus erythematosus, toxic shock syndrome, viral, bacterial, fungal, helminthic and protozoal infections.

ADMINISTRATION - The composition is administered orally, intranasally, anally, topically or by injection (claimed), parenterally (including intravenously, intraarterially, subcutaneously, intraperitoneally, intracranially, intramuscularly or by infusion), intrathecally, intraventricularly, locally, systemically, vaginally, rectally, pulmonary, by inhalation, as aerosol, intranasally, epidermally, transdermally, as liposome or ophthalmically in a dosage of 0.01 ug - 100 g.

ADVANTAGE - The anti-sense oligonucleotide inhibits expression of **human kinesin** gene such as **CENP-E** having nucleic acid sequence deposited in GenBank as GenBank ID Z15005, **human** Eg5 having nucleic acid sequence deposited in GenBank as GenBank ID U37426 and MCAK gene having nucleic acid sequence deposited in GenBank as GenBank ID U63743.

EXAMPLE - No relevant example given. (29 pages)

L20 ANSWER 6 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2002:950597 SCISEARCH
THE GENUINE ARTICLE: 615PA
TITLE: The mitotic-spindle-associated protein astrin is essential for progression through mitosis
AUTHOR: Gruber J; Harborth J; Schnabel J; Weber K; Hatzfeld M (Reprint)
CORPORATE SOURCE: Univ Halle Wittenberg, Fac Med, Dept Biochem & Pathobiochem, D-06097 Halle Saale, Germany (Reprint); Max Planck Inst Biophys Chem, Dept Biochem, D-37070 Gottingen, Germany
COUNTRY OF AUTHOR: Germany
SOURCE: JOURNAL OF CELL SCIENCE, (1 NOV 2002) Vol. 115, No. 21, pp. 4053-4059.
Publisher: COMPANY OF BIOLOGISTS LTD, BIDDER BUILDING CAMBRIDGE COMMERCIAL PARK COWLEY RD, CAMBRIDGE CB4 4DL, CAMBS, ENGLAND.
ISSN: 0021-9533.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 41

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Astrin is a mitotic-spindle-associated protein expressed in most **human** cell lines and tissues. However, its functions in spindle organization and mitosis have not yet been determined. Sequence analysis revealed that astrin has an N-terminal globular domain and an extended coiled-coil domain. Recombinant astrin was purified and characterized by CD spectroscopy and electron microscopy. Astrin showed parallel dimers with head-stalk structures reminiscent of **motor** proteins, although no sequence similarities to known **motor** proteins were found. In physiological buffers, astrin dimers oligomerized via their globular head domains and formed aster-like structures. Silencing of astrin in HeLa cells by RNA interference resulted in growth arrest, with formation of multipolar and highly disordered spindles. Chromosomes did not congress to the spindle equator and remained dispersed. Cells depleted of astrin were normal during interphase but were unable to progress through mitosis and finally ended in apoptotic cell death. Possible functions of astrin in mitotic spindle organization are discussed.

L20 ANSWER 7 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 2002:978474 SCISEARCH

THE GENUINE ARTICLE: 621KX
TITLE: Protein kinase TTK interacts and co-localizes with **CENP-E** to the kinetochore of **human** cells
AUTHOR: Zhang J; Fu C H; Miao Y; Dou Z; Yao X B (Reprint)
CORPORATE SOURCE: Univ Sci & Technol China, Lab Cell Dynam, Hefei 230027, Peoples R China (Reprint)
COUNTRY OF AUTHOR: Peoples R China
SOURCE: CHINESE SCIENCE BULLETIN, (DEC 2002) Vol. 47, No. 23, pp. 2005-2009.
Publisher: SCIENCE CHINA PRESS, 16 DONGHUANGCHENGGEN NORTH ST, BEIJING 100717, PEOPLES R CHINA.
ISSN: 1001-6538.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 25

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Spindle checkpoint is an important biochemical signaling cascade during mitosis which monitors the fidelity of chromosome segregation, and is mediated by protein kinases Mps1 and Bub1/BubR1. Our recent studies show that **kinesin**-related **motor** protein **CENP-E** interacts with BubR1 and participates in spindle checkpoint signaling. To elucidate the molecular mechanisms underlying spindle checkpoint signaling, we carried out proteomic dissection of **human** cell kinetochore and revealed protein kinase TTK, **human** homologue of yeast Mps1. Our studies show that TTK is, localized to the kinetochore of **human** cells, and interacts with **CENP-E**, suggesting that TTK may play an important role in chromosome segregation during mitosis.

L20 ANSWER 8 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2002:174077 SCISEARCH
THE GENUINE ARTICLE: 520WH
TITLE: Zebrafish mitotic **kinesin**-like protein 1 (Mklp1) functions in embryonic cytokinesis
AUTHOR: Chen M C; Zhou Y; Detrich H W (Reprint)
CORPORATE SOURCE: Northeastern Univ, Dept Biol, 414 Mugar Hall, 360 Huntington Ave, Boston, MA 02115 USA (Reprint); Northeastern Univ, Dept Biol, Boston, MA 02115 USA; Childrens Hosp, Div Hematol Oncol, Boston, MA 02115 USA; Howard Hughes Med Inst, Boston, MA 02115 USA
COUNTRY OF AUTHOR: USA
SOURCE: PHYSIOLOGICAL GENOMICS, (11 FEB 2002) Vol. 8, No. 1, pp. 51-66.
Publisher: AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814 USA.
ISSN: 1094-8341.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 76

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB To understand the functions of microtubule **motors** in vertebrate development, we are investigating the **kinesin**-like proteins (KLPs) of the zebrafish, Danio rerio. Here we describe the structure, intracellular distribution, and function of zebrafish mitotic KLP1 (Mklp1). The zebrafish mklp1 gene that encodes this 867-amino acid protein maps to a region of zebrafish linkage group 18 that is syntenic with part of **human** chromosome 15. In zebrafish AB9 fibroblasts and in COS-7 cells, the zebrafish Mklp1 protein decorates spindle microtubules at metaphase, redistributes to the spindle midzone during anaphase, and becomes concentrated in the midbody during telophase and cytokinesis. The **motor** is detected consistently in interphase nuclei of COS cells and occasionally in those of AB9 cells. Nuclear

targeting of Mklp1 is conferred by two basic motifs located in the COOH terminus of the **motor**. In cleaving zebrafish embryos, green fluorescent protein (GFP)-tagged Mklp1 is found in the nucleus in interphase and associates with microtubules of the spindle midbody in cytokinesis. One- or two-cell embryos injected with synthetic mRNAs encoding dominant-negative variants of GFP-Mklp1 frequently fail to complete cytokinesis during cleavage, resulting in formation of multinucleated blastomeres. Our results indicate that the zebrafish Mklp1 **motor** performs a critical function that is required for completion of embryonic cytokinesis.

L20 ANSWER 9 OF 34 MEDLINE on STN
ACCESSION NUMBER: 2001688509 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11734897
TITLE: Maximum likelihood methods reveal conservation of function among closely related **kinesin** families.
AUTHOR: Lawrence Carolyn J; Malmberg Russell L; Muszynski Michael G; Dawe R Kelly
CORPORATE SOURCE: University of Georgia, Department of Botany, Athens, GA 30602, USA.
SOURCE: Journal of molecular evolution, (2002 Jan) 54 (1) 42-53. Journal code: 0360051. ISSN: 0022-2844.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 20011206
Last Updated on STN: 20020816
Entered Medline: 20020815

AB We have reconstructed the evolution of the anciently derived **kinesin** superfamily using various alignment and tree-building methods. In addition to classifying previously described **kinesins** from protists, fungi, and animals, we analyzed a variety of **kinesin** sequences from the plant kingdom including 12 from Zea mays and 29 from Arabidopsis thaliana. Also included in our data set were four sequences from the anciently diverged amitochondriate protist Giardia lamblia. The overall topology of the best tree we found is more likely than previously reported topologies and allows us to make the following new observations: (1) **kinesins** involved in chromosome movement including MCAK, chromokinesin, and **CENP-E** may be descended from a single ancestor; (2) **kinesins** that form complex oligomers are limited to a monophyletic group of families; (3) **kinesins** that crosslink antiparallel microtubules at the spindle midzone including BIMC, MKLP, and **CENP-E** are closely related; (4) Drosophila NOD and **human** KID group with other characterized chromokinesins; and (5) Saccharomyces SMY1 groups with **kinesin**-I sequences, forming a family of **kinesins** capable of class V myosin interactions. In addition, we found that one monophyletic clade composed exclusively of sequences with a C-terminal **motor** domain contains all known minus end-directed **kinesins**.

L20 ANSWER 10 OF 34 MEDLINE on STN
ACCESSION NUMBER: 2001417117 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11382767
TITLE: Purification and characterization of native conventional **kinesin**, HSET, and **CENP-E** from mitotic hela cells.
AUTHOR: DeLuca J G; Newton C N; Himes R H; Jordan M A; Wilson L
CORPORATE SOURCE: Department of Molecular, Cellular, and Developmental Biology and the Materials Research Laboratory, University of California, Santa Barbara, California 93106, USA.
CONTRACT NUMBER: CA57291 (NCI)

NS13560 (NINDS)
SOURCE: Journal of biological chemistry, (2001 Jul 27) 276 (30) 28014-21.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200108
ENTRY DATE: Entered STN: 20010827
Last Updated on STN: 20030105
Entered Medline: 20010823

AB We have developed a strategy for the purification of native microtubule **motor** proteins from mitotic HeLa cells and describe here the purification and characterization of **human** conventional **kinesin** and two **human kinesin**-related proteins, HSET and **CENP-E**. We found that the 120-kDa HeLa cell conventional **kinesin** is an active **motor** that induces microtubule gliding at approximately 30 microm/min at room temperature. This active form of HeLa cell **kinesin** does not contain light chains, although light chains were detected in other fractions. HSET, a member of the C-terminal **kinesin** subfamily, was also purified in native form for the first time, and the protein migrates as a single band at approximately 75 kDa. The purified HSET is an active **motor** that induces microtubule gliding at a rate of approximately 5 microm/min, and microtubules glide for an average of 3 microm before ceasing movement. Finally, we purified native **CENP-E**, a **kinesin**-related protein that has been implicated in chromosome congression during mitosis, and we found that this form of **CENP-E** does not induce microtubule gliding but is able to bind to microtubules.

L20 ANSWER 11 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 2001:165210 SCISEARCH
THE GENUINE ARTICLE: 400QK
TITLE: Chromosome movement in mitosis requires microtubule anchorage at spindle poles
AUTHOR: Gordon M B; Howard L; Compton D A (Reprint)
CORPORATE SOURCE: Dartmouth Med Sch, Dept Biochem, Hanover, NH 03755 USA (Reprint); Dartmouth Coll, Rippel Electron Microscope Facil, Hanover, NH 03755 USA
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CELL BIOLOGY, (5 FEB 2001) Vol. 152, No. 3, pp. 425-434.
Publisher: ROCKEFELLER UNIV PRESS, 1114 FIRST AVE, 4TH FL, NEW YORK, NY 10021 USA.
ISSN: 0021-9525.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 76
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Anchorage of microtubule minus ends at spindle poles has been proposed to bear the load of poleward forces exerted by kinetochore-associated **motors** so that chromosomes move toward the poles rather than the poles toward the chromosomes. To test this hypothesis, we monitored chromosome movement during mitosis after perturbation of nuclear mitotic apparatus protein (NuMA) and the **human** homologue of the KIN C **motor** family (HSET), two noncentrosomal proteins involved in spindle pole organization in animal cells. Perturbation of NuMA alone disrupts spindle pole organization and delays anaphase onset, but does not alter the velocity of oscillatory chromosome movement in prometaphase. Perturbation of HSET alone increases the duration of prometaphase, but does not alter the velocity of chromosome movement in prometaphase or

anaphase. In contrast, simultaneous perturbation of both HSET and NuMA severely suppresses directed chromosome movement in prometaphase. Chromosomes coalesce near the center of these cells on bi-oriented spindles that lack organized poles. Immunofluorescence and electron microscopy verify microtubule attachment to sister kinetochores, but this attachment fails to generate proper tension across sister kinetochores. These results demonstrate that anchorage of microtubule minus ends at spindle poles mediated by overlapping mechanisms involving both NuMA and HSET is essential for chromosome movement during mitosis.

L20 ANSWER 12 OF 34 MEDLINE on STN
ACCESSION NUMBER: 2001338615 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11250166
TITLE: Chromosome movement: dynein-out at the kinetochore.
AUTHOR: Banks J D; Heald R
CORPORATE SOURCE: Department of Molecular and Cell Biology, University of California, Berkeley, California 94720-3200, USA.. jenbanks@uclink4.berkeley.edu
SOURCE: Current biology : CB, (2001 Feb 20) 11 (4) R128-31. Ref: 28
Journal code: 9107782. ISSN: 0960-9822.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200106
ENTRY DATE: Entered STN: 20010618
Last Updated on STN: 20010618
Entered Medline: 20010614
AB Cell biologists have long speculated that a minus end-directed **motor** localized at kinetochores contributes to the poleward movement of chromosomes during mitosis. Two recent studies provide direct evidence that cytoplasmic dynein can perform this function.

L20 ANSWER 13 OF 34 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:756837 HCAPLUS
DOCUMENT NUMBER: 133:318271
TITLE: Recombinant bacterial expression and purification of human kinesins
INVENTOR(S): Beraud, Christophe; Ohashi, Cara; **Sakowicz, Roman**; Wood, Ken; Vaisberg, Eugeni; Yu, Ming
PATENT ASSIGNEE(S): Cytokinetics, USA
SOURCE: PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000063353	A1	20001026	WO 2000-US10870	20000420
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6544766	B1	20030408	US 2000-595684	20000616

US 6387644	B1	20020514	US 2000-724224	20001128
US 2003044900	A1	20030306	US 2001-45631	20011019
US 6762043	B1	20040713	US 2002-93317	20020306
US 2004142397	A1	20040722	US 2004-797893	20040309

PRIORITY APPLN. INFO.:

US 1999-295612	A1	19990420
WO 2000-US10870	A1	20000420
US 2000-597292	B1	20000620
US 2000-724224	A1	20001128
US 2002-93317	A3	20020306

AB Described herein are methods of producing kinesins. In a preferred embodiment, the kinesins are produced from a prokaryote, most preferably, a bacterial cell. Bacterial expression offers several advantages over systems previously utilized, such as, for example, Baculovirus. The yield of protein is higher, the cost of the expression setup is lower, and creation of alternative expression vectors is easier. The concern of copurifying a contaminating activity from the expression host is also eliminated since bacteria, in contrast to the baculovirus expression system, do not have kinesin-like proteins. Also described herein are purified kinesins, preferably unglycosylated and methods of use.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 14 OF 34 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:194248 HCAPLUS
DOCUMENT NUMBER: 130:233824
TITLE: Plus end-directed microtubule motor protein **CENP-E** required for Xenopus chromosome congression
INVENTOR(S): Wood, Kenneth W.; **Sakowicz, Roman**; Goldstein, Lawrence S. B.; Cleveland, Don W.
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 78 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9913061	A1	19990318	WO 1998-US19231	19980910
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2303484	AA	19990318	CA 1998-2303484	19980910
AU 9893918	A1	19990329	AU 1998-93918	19980910
AU 745385	B2	20020321		
EP 1012249	A1	20000628	EP 1998-947039	19980910
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001526881	T2	20011225	JP 2000-510850	19980910
US 6645748	B1	20031111	US 1998-150867	19980910

PRIORITY APPLN. INFO.:

US 1997-58645P	P	19970911
WO 1998-US19231	W	19980910

AB The invention provides isolated nucleic acid and amino acid sequences of Xenopus centromere-associated protein-E (XCENP-E), antibodies to XCENP-E, methods of screening for **CENP-E** modulators using biol. active **CENP-E**, and kits for screening for **CENP -E** modulators. The full-length cDNA sequences of XCENP-E

encodes a protein of 2954 amino acids with a predicted mol. mass of 340 kDa. XCENP-E is a member of the kinesin superfamily of motor proteins, and consists of a 500-amino acid globular N-terminal domain containing a kinesin-like microtubule motor domain linked to a globular tail domain by a region predicted to form a long, discontinuous α-helical coiled coil. The is the first biol. active **CENP-E** isolated and, surprisingly and contrary to previous reports, it demonstrates a motor that powers chromosome movement toward microtubule plus ends. Using immunodepletion and antibody addition to Xenopus egg exts., the present invention further demonstrates that **CENP-E** plays an essential role in congression.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 15 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 1999:980179 SCISEARCH
THE GENUINE ARTICLE: 255MW
TITLE: The role of the kinetochore protein **CENP-E** in the mitotic checkpoint in xenopus egg extract.
AUTHOR: Abrieu A (Reprint); **Wood K**; Kahana J; Cleveland D W
CORPORATE SOURCE: LUDWIG INST CANC RES, LA JOLLA, CA 92093
COUNTRY OF AUTHOR: USA
SOURCE: MOLECULAR BIOLOGY OF THE CELL, (NOV 1999) Vol. 10, Supp. [S], pp. 730-730.
Publisher: AMER SOC CELL BIOLOGY, PUBL OFFICE, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.
ISSN: 1059-1524.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 0

L20 ANSWER 16 OF 34 MEDLINE on STN

ACCESSION NUMBER: 1998167852 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9499420
TITLE: Localization of **motor**-related proteins and associated complexes to active, but not inactive, centromeres.
AUTHOR: Faulkner N E; Vig B; Echeverri C J; Wordeman L; Vallee R B
CORPORATE SOURCE: Cell Biology Group, Worcester Foundation for Biomedical Research, Shrewsbury, MA 01545, USA.
CONTRACT NUMBER: GM478434 (NIGMS)
SOURCE: Human molecular genetics, (1998 Apr) 7 (4) 671-7.
Journal code: 9208958. ISSN: 0964-6906.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 19980520
Last Updated on STN: 19980520
Entered Medline: 19980512

AB Multicentric chromosomes are often found in tumor cells and certain cell lines. How they are generated is not fully understood, though their stability suggests that they are non-functional during chromosome segregation. Growing evidence has implicated microtubule **motor** proteins in attachment of chromosomes to the mitotic spindle and in chromosome movement. To better understand the molecular basis for the inactivity of centromeres associated with secondary constrictions, we have tested these structures by immunofluorescence microscopy for the presence of **motor** complexes and associated proteins. We find strong

immunoreactivity at the active, but not inactive, centromeres of prometaphase multicentric chromosomes using antibodies to the cytoplasmic dynein intermediate chains, three components of the dynactin complex (dynamitin, Arp1 and p150 Glued), the **kinesin**-related proteins **CENP-E** and MCAK and the proposed structural and checkpoint proteins HZW10, CENP-F and Mad2p. These results offer new insight into the assembly and composition of both primary and secondary constrictions and provide a molecular basis for the apparent inactivity of the latter during chromosome segregation.

L20 ANSWER 17 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 1998:437989 SCISEARCH
THE GENUINE ARTICLE: ZR489
TITLE: Rigor-type mutation in the **kinesin**-related protein HsEg5 changes its subcellular localization and induces microtubule bundling
AUTHOR: Blangy A (Reprint); Chaussepied P; Nigg E A
CORPORATE SOURCE: CNRS, CRBM, IFR 24, 1919 ROUTE MENDE, F-34033 MONTPELLIER, FRANCE (Reprint); SWISS INST EXPT CANC RES, CH-1066 EPALINGES, SWITZERLAND; UNIV GENEVA, DEPT MOL BIOL, CH-1211 GENEVA, SWITZERLAND
COUNTRY OF AUTHOR: FRANCE; SWITZERLAND
SOURCE: CELL MOTILITY AND THE CYTOSKELETON, (FEB 1998) Vol. 40, No. 2, pp. 174-182.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0886-1544.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 54

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB HsEg5 is a **human kinesin**-related **motor** protein essential for the formation of a bipolar mitotic spindle. It interacts with the mitotic centrosomes in a phosphorylation-dependent manner. To investigate further the mechanisms involved in targetting HsEg5 to the spindle apparatus, we expressed various mutants of HsEg5 in HeLa cells. All these mutants share a mutation of Thr-112 in the N-terminal **motor** domain, resulting in the inactivation of the ATP binding domain. In vitro, the HsEg5-T112N mutant **motor** domain showed a nucleotide-independent microtubule association, typical of a **kinesin** protein binding to microtubules in a rigor state. In vivo, overexpression of the HsEg5 rigor mutant in HeLa cells induced, in interphase, microtubule bundling, and, in mitosis, the formation of monopolar mitotic spindles similar to those observed after microinjection of anti-HsEg5 antibodies. Localization of the HsEg5 rigor mutant on cytoplasmic microtubules did not require the C-terminal tail domain but was lost when the stalk domain was also deleted. Sucrose gradient centrifugation experiments showed that microtubule bundling was most likely caused by the binding of HsEg5 mutants in a dimeric state. These results demonstrate that the precise subcellular localization of HsEg5 in vivo is regulated not only by the phosphorylation of the tail domain but also by the oligomeric state of the protein. (C) 1998 Wiley-Liss, Inc.

L20 ANSWER 18 OF 34 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 97258096 EMBASE
DOCUMENT NUMBER: 1997258096
TITLE: **CENP-E** is an essential kinetochore **motor** in maturing oocytes and is masked during mos-dependent, cell cycle arrest at metaphase II.
AUTHOR: Duesbery N.S.; Choi T.; Brown K.D.; Wood K.W.; Resau J.; Fukasawa K.; Cleveland D.W.; Vande Woude G.F.

CORPORATE SOURCE: G.F. Vande Woude, ABL-Basic Research Program, National Cancer Institute, Frederick Cancer Res./Devt. Center, P.O. Box B, Frederick, MD 21702-1201, United States
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997) 94/17 (9165-9170). Refs: 53
ISSN: 0027-8424 CODEN: PNASA6
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 021 Developmental Biology and Teratology
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

AB **CENP-E**, a **kinesin**-like protein that is known to associate with kinetochores during all phases of mitotic chromosome movement, is shown here to be a component of meiotic kinetochores as well. **CENP-E** is detected at kinetochores during metaphase I in both mice and frogs, and, as in mitosis, is relocalized to the midbody during telophase. **CENP-E** function is essential for meiosis I because injection of an antibody to **CENP-E** into mouse oocytes in prophase completely prevented progression of those oocytes past metaphase I. Beyond this, **CENP-E** is modified or masked during the natural, Mos- dependent, cell cycle arrest that occurs at metaphase II, although it is readily detectable at the kinetochores in metaphase II oocytes derived from mos-deficient (MOS(-/-)) mice that fail to arrest at metaphase II. This must reflect a masking of some **CENP-E** epitopes, not the absence of **CENP-E**, in meiosis II because a different polyclonal antibody raised to the tail of **CENP-E** detects **CENP-E** at kinetochores of metaphase II-arrested eggs and because **CENP-E** reappears in telophase of mouse oocytes activated in the absence of protein synthesis.

L20 ANSWER 19 OF 34 MEDLINE on STN
ACCESSION NUMBER: 97361828 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9218789
TITLE: Identification of a **motor** protein required for filamentous growth in Ustilago maydis.
AUTHOR: Lehmler C; Steinberg G; Snetselaar K M; Schliwa M; Kahmann R; Bolker M
CORPORATE SOURCE: Institut fur Genetik und Mikrobiologie der Universitat Munchen, Germany.
SOURCE: EMBO journal, (1997 Jun) 16 (12) 3464-73.
Journal code: 8208664. ISSN: 0261-4189.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L47106; GENBANK-U92844; GENBANK-U92845; SWISSPROT-Q02224
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970825
Last Updated on STN: 20030228
Entered Medline: 19970808

AB The phytopathogenic fungus Ustilago maydis exists in two stages, the yeast-like haploid form and the filamentous dikaryon. Both pathogenicity and dimorphism are genetically controlled by two mating-type loci, with only the filamentous stage being pathogenic on corn. We have identified two genes (kin1 and kin2) encoding **motor** proteins of the **kinesin** family. Kin1 is most similar to the **human** **CENP-E** gene product, while Kin2 is most closely related to the conventional **kinesin** Nkin of Neurospora crassa. Deletion mutants of kin1 had no discernible phenotype; delta kin2 mutants, however, were severely affected in hyphal extension and pathogenicity. The

wild-type dikaryon showed rapid tip growth, with all the cytoplasm being moved to the tip compartment. Left behind are septate cell wall tubes devoid of cytoplasm. In delta kin2 mutants, dikaryotic cells were formed after cell fusion, but these hyphal structures remained short and filled with cytoplasm. A functional green fluorescent protein (GFP)-Kin2 fusion was generated and used to determine the localization of the **motor** protein by fluorescence microscopy. Inspection of the hyphal tips by electron microscopy revealed a characteristic accumulation of darkly stained vesicles which was absent in mutant cells. We suggest that the **motor** protein Kin2 is involved in organizing this specialized growth zone at the hyphal tip, probably by affecting the vectorial transport of vesicles.

L20 ANSWER 20 OF 34 MEDLINE on STN
ACCESSION NUMBER: 1998060834 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9396744
TITLE: **CENP-E** function at kinetochores is essential for chromosome alignment.
AUTHOR: Schaar B T; Chan G K; Maddox P; Salmon E D; Yen T J
CORPORATE SOURCE: Cell and Molecular Biology Graduate Group, University of Pennsylvania, Philadelphia, Pennsylvania 19103, USA.
CONTRACT NUMBER: CA06927 (NCI)
GM24364 (NIGMS)
GM44762 (NIGMS)
SOURCE: Journal of cell biology, (1997 Dec 15) 139 (6) 1373-82.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980129
Last Updated on STN: 19980129
Entered Medline: 19980113

AB **CENP-E** is a **kinesin**-like protein that binds to kinetochores and may provide functions that are critical for normal chromosome motility during mitosis. To directly test the in vivo function of **CENP-E**, we microinjected affinity-purified antibodies to block the assembly of **CENP-E** onto kinetochores and then examined the behavior of these chromosomes. Chromosomes lacking **CENP-E** at their kinetochores consistently exhibited two types of defects that blocked their alignment at the spindle equator. Chromosomes positioned near a pole remained mono-oriented as they were unable to establish bipolar microtubule connections with the opposite pole. Chromosomes within the spindle established bipolar connections that supported oscillations and normal velocities of kinetochore movement between the poles, but these bipolar connections were defective because they failed to align the chromosomes into a metaphase plate. Overexpression of a mutant that lacked the amino-terminal 803 amino acids of **CENP-E** was found to saturate limiting binding sites on kinetochores and competitively blocked endogenous **CENP-E** from assembling onto kinetochores. Chromosomes saturated with the truncated **CENP-E** mutant were never found to be aligned but accumulated at the poles or were strewn within the spindle as was the case when cells were microinjected with **CENP-E** antibodies. As the **motor** domain was contained within the portion of **CENP-E** that was deleted, the chromosomal defect is likely attributed to the loss of **motor** function. The combined data show that **CENP-E** provides kinetochore functions that are essential for monopolar chromosomes to establish bipolar connections and for chromosomes with connections to both spindle poles to align at the spindle equator. Both of these events rely on activities that are provided by **CENP-E's motor** domain.

L20 ANSWER 21 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 1998:25462 SCISEARCH
THE GENUINE ARTICLE: YM669
TITLE: Localization of **CENP-E** in the fibrous corona and outer plate of mammalian kinetochores from prometaphase through anaphase
AUTHOR: Cooke C A; Schaar B; Yen T J; Earnshaw W C (Reprint)
CORPORATE SOURCE: UNIV EDINBURGH, INST CELL & MOL BIOL, MICHAEL SWANN BLDG, KINGS BLDG, MAYFIELD RD, EDINBURGH EH9 3JR, MIDLOTHIAN, SCOTLAND (Reprint); UNIV EDINBURGH, INST CELL & MOL BIOL, EDINBURGH EH9 3JR, MIDLOTHIAN, SCOTLAND; FOX CHASE CANC CTR, PHILADELPHIA, PA 19111
COUNTRY OF AUTHOR: SCOTLAND; USA
SOURCE: CHROMOSOMA, (1 DEC 1997) Vol. 106, No. 7, pp. 446-455. Publisher: SPRINGER VERLAG, 175 FIFTH AVE, NEW YORK, NY 10010.
ISSN: 0009-5915.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 22
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB We have conducted a detailed ultrastructural analysis of the distribution of the **kinesin**-related centromere protein **CENP-E** during mitosis in cultured **human**, rat kangaroo and Indian muntjac cells. Using an affinity-purified polyclonal antibody and detection by 0.8 nm colloidal gold particles, **CENP-E** was localized primarily to the fibrous corona of the kinetochore in prometaphase and metaphase cells. Some labeling of the kinetochore outer plate was also observed. The distribution of fibrous corona-associated **CENP-E** did not change dramatically following the attachment of microtubules to the kinetochore. Thus, the normal disappearance of this kinetochore substructure in conventional electron micrographs of mitotic chromosomes with attached kinetochores is not due to the corona becoming stretched along the spindle microtubules as has been suggested. Examination of cells undergoing anaphase chromatid movement revealed the presence of **CENP-E** still associated with the outer surface of the kinetochore plate. At the same time, the majority of detectable **CENP-E** in these cells was associated with the bundles of antiparallel microtubules in the central spindle. **CENP-E** in this region of the cell is apparently associated with the stem body matrix material. The simultaneous localization of **CENP-E** on centromeres and the central spindle during anaphase was confirmed by both wide-field microscopy of **human** cells and conventional fluorescence microscopy of rat kangaroo cells. Together, the observations reported here are consistent with models in which **CENP-E** has a role in promoting the poleward migration of sister chromatids during anaphase A.

L20 ANSWER 22 OF 34 MEDLINE on STN
ACCESSION NUMBER: 97477390 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9334346
TITLE: The microtubule-dependent **motor** centromere-associated protein E (**CENP-E**) is an integral component of kinetochore corona fibers that link centromeres to spindle microtubules.
AUTHOR: Yao X; Anderson K L; Cleveland D W
CORPORATE SOURCE: Laboratory of Cell Biology, Ludwig Institute for Cancer Research, School of Medicine, University of California, La Jolla, CA 92093-0660, USA.
CONTRACT NUMBER: GM 29513 (NIGMS)
SOURCE: Journal of cell biology, (1997 Oct 20) 139 (2) 435-47.

Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199711
ENTRY DATE: Entered STN: 19971224
Last Updated on STN: 19971224
Entered Medline: 19971120

AB Centromere-associated protein E (**CENP-E**) is a **kinesin**-related microtubule **motor** protein that is essential for chromosome congression during mitosis. Using immunoelectron microscopy, **CENP-E** is shown to be an integral component of the kinetochore corona fibers that tether centromeres to the spindle. Immediately upon nuclear envelope fragmentation, an associated plus end **motor** trafficks cytoplasmic **CENP-E** toward chromosomes along astral microtubules that enter the nuclear volume. Before or concurrently with initial lateral attachment of spindle microtubules, **CENP-E** targets to the outermost region of the developing kinetochores. After stable attachment, throughout chromosome congression, at metaphase, and throughout anaphase A, **CENP-E** is a constituent of the corona fibers, extending at least 50 nm away from the kinetochore outer plate and intertwining with spindle microtubules. In congressing chromosomes, **CENP-E** is preferentially associated with (or accessible at) the stretched, leading kinetochore known to provide the primary power for chromosome movement. Taken together, this evidence strongly supports a model in which **CENP-E** functions in congression to tether kinetochores to the disassembling microtubule plus ends.

L20 ANSWER 23 OF 34 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 1998028574 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9363944
TITLE: **CENP-E** is a plus end-directed kinetochore **motor** required for metaphase chromosome alignment.
AUTHOR: Wood K W; Sakowicz R; Goldstein L S; Cleveland D W
CORPORATE SOURCE: Laboratory of Cell Biology, Ludwig Institute for Cancer Research, University of California at San Diego, La Jolla 92093-0660, USA.
SOURCE: Cell, (1997 Oct 31) 91 (3) 357-66.
Journal code: 0413066. ISSN: 0092-8674.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF027728
ENTRY MONTH: 199712
ENTRY DATE: Entered STN: 19980109
Last Updated on STN: 19980109
Entered Medline: 19971210

AB Mitosis requires dynamic attachment of chromosomes to spindle microtubules. This interaction is mediated largely by kinetochores. During prometaphase, forces exerted at kinetochores, in combination with polar ejection forces, drive congression of chromosomes to the metaphase plate. A major question has been whether kinetochore-associated microtubule **motors** play an important role in congression. Using immunodepletion from and antibody addition to Xenopus egg extracts, we show that the kinetochore-associated **kinesin**-like **motor** protein **CENP-E** is essential for positioning chromosomes at the metaphase plate. We further demonstrate that **CENP-E** powers movement toward microtubule plus ends in vitro. These findings support a model in which **CENP-E** functions in congression to tether kinetochores to dynamic microtubule

plus ends.

L20 ANSWER 24 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 97:370707 SCISEARCH
THE GENUINE ARTICLE: WX609
TITLE: Increased chromokinesin immunoreactivity in retinoblastoma cells
AUTHOR: Yan R T; Wang S Z (Reprint)
CORPORATE SOURCE: UNIV ALABAMA, SCH MED, EYE FDN HOSP, DEPT OPHTHALMOL, 700 S 18TH ST, BIRMINGHAM, AL 35233 (Reprint); UNIV ALABAMA, SCH MED, EYE FDN HOSP, DEPT OPHTHALMOL, BIRMINGHAM, AL 35233
COUNTRY OF AUTHOR: USA
SOURCE: GENE, (21 APR 1997) Vol. 189, No. 2, pp. 263-267. Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS. ISSN: 0378-1119.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 17

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB chromokinesin is a developmentally down-regulated gene with specific expression in proliferating cells during embryonic chick development. It encodes a DNA-binding **motor** protein localized along the chromosome arm during mitosis, suggesting that the protein may be a component of the long-observed, yet poorly understood 'ejection force' hypothesized to be involved in controlling the direction and speed of chromosome movement. We have isolated **human** chromokinesin; with affinity-purified antibodies we demonstrated immunocytochemically that Chromokinesin was present at a much higher level in cultured retinoblastoma cells than in primary cultures of **human** dermal fibroblasts. The increase in immunoreactivity was particularly prominent in interphase cells, whereas in primary cultures of fibroblasts immunopositive cells were predominantly M-phase cells. These observations imply a deregulation of chromokinesin in retinoblastoma cells. Data presented here may be useful in designing strategies to modulate chromosome movement and cell proliferation with either antisense oligonucleotides or specific antibodies, and hence may set the stage for further investigations of the involvement of chromosome **motor** molecules in mitosis under normal and pathological conditions. (C) 1997 Elsevier Science B.V.

L20 ANSWER 25 OF 34 MEDLINE on STN
ACCESSION NUMBER: 96338605 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8743943
TITLE: The **kinesin**-like protein **CENP-E** is kinetochore-associated throughout poleward chromosome segregation during anaphase-A.
AUTHOR: Brown K D; Wood K W; Cleveland D W
CORPORATE SOURCE: Department of Biological Chemistry, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA.
CONTRACT NUMBER: GM 29513 (NIGMS)
SOURCE: Journal of cell science, (1996 May) 109 (Pt 5) 961-9. Journal code: 0052457. ISSN: 0021-9533.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199612
ENTRY DATE: Entered STN: 19970128
Last Updated on STN: 19970128
Entered Medline: 19961231

AB The **kinesin**-like protein **CENP-E** transiently associates with kinetochores following nuclear envelope breakdown in late prophase, remains bound throughout metaphase, but sometime after anaphase onset it releases and by telophase becomes bound to interzonal microtubules of the mitotic spindle. Inhibition of poleward chromosome movement in vitro by **CENP-E** antibodies and association of **CENP-E** with minus-end directed microtubule motility in vitro have combined to suggest a key role for **CENP-E** as an anaphase chromosome **motor**. For this to be plausible in vivo depends on whether **CENP-E** remains kinetochore associated during anaphase. Using Indian muntjac cells whose seven chromosomes have large, easily tracked kinetochores, we now show that **CENP-E** is kinetochore-associated throughout the entirety of anaphase-A (poleward chromosome movement), relocating gradually during spindle elongation (anaphase-B) to the interzonal microtubules. These observations support roles for **CENP-E** not only in the initial alignment of chromosomes at metaphase and in spindle elongation in anaphase-B, but also in poleward chromosome movement in anaphase-A.

L20 ANSWER 26 OF 34 BIOSIS COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 1996:524605 BIOSIS
DOCUMENT NUMBER: PREV199699246961
TITLE: Modulation of **CENP-E** organization at kinetochores by spindle microtubule attachment.
AUTHOR(S): Thrower, Douglas A. [Reprint author]; Jordan, Mary Ann; Wilson, Leslie
CORPORATE SOURCE: Dep. Mol., Cell. Dev. Biol., Univ. Calif., Santa Barbara, CA 93106, USA
SOURCE: Cell Motility and the Cytoskeleton, (1996) Vol. 35, No. 2, pp. 121-133.
CODEN: CMCYEO. ISSN: 0886-1544.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 Nov 1996
Last Updated on STN: 22 Nov 1996

AB **CENP-E** is a protein of the **kinesin** superfamily that appears as small paired globules at kinetochores of chromosomes in mammalial cells during prometaphase and metaphase of mitosis (Yen et al., 1992: Nature 359:536-539). In the present study we found that a significant number of chromosomes during early prometaphase in HeLa cells (approximately 30%) were stained with a **CENP-E** antibody in the form of large C-shaped "collars" that partially encircled the chromosomes. The C-shaped **CENP-E** collars were present only transiently and were completely replaced by small paired globular forms prior to metaphase. Most chromosomes had persistent **CENP-E** collars in cells blocked at mitosis with a vinblastine concentration sufficient to prevent all microtubule formation. Attachment of newly formed microtubules to the kinetochores after removal of vinblastine resulted in loss of the collars and replacement with small paired globules. Similarly, a higher proportion of chromosomes isolated from vinblastine-treated cells contained **CENP-E** collars (73%), and the "capture" (i.e., attachment) of microtubules by the chromosomes resulted in conversion of the collars into small paired globules in vitro. Thus, the **CENP-E** collars form prior to microtubule attachment and disappear after attachment of the chromosomes to the spindle. The **CENP-E** collars may facilitate capture of microtubules by chromosomes during prometaphase.

L20 ANSWER 27 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN

ACCESSION NUMBER: 95:329093 SCISEARCH
THE GENUINE ARTICLE: QY118

TITLE: CHARACTERIZATION OF A MINUS END-DIRECTED **KINESIN** -LIKE **MOTOR** PROTEIN FROM CULTURED-MAMMALIAN-CELLS

AUTHOR: KURIYAMA R (Reprint); KOFRON M; ESSNER R; KATO T; DRAGASGRANOIC S; OMOTO C K; KHODJAKOV A

CORPORATE SOURCE: UNIV MINNESOTA, DEPT CELL BIOL & NEUROANAT, 4-135 JACKSON HALL, 321 CHURCH ST SE, MINNEAPOLIS, MN, 55455 (Reprint); WASHINGTON STATE UNIV, DEPT GENET & CELL BIOL, PULLMAN, WA, 99164

COUNTRY OF AUTHOR: USA

SOURCE: JOURNAL OF CELL BIOLOGY, (MAY 1995) Vol. 129, No. 4, pp. 1049-1059.
ISSN: 0021-9525.

DOCUMENT TYPE: Article; Journal

FILE SEGMENT: LIFE

LANGUAGE: ENGLISH

REFERENCE COUNT: 51

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Using the CHO2 monoclonal antibody raised against CHO spindles (Sellitto, C., M. Kimble, and R. Kuriyama. 1992. Cell Motil. Cytoskeleton. 22:7-24) we identified a 66-kD protein located at the interphase centrosome and mitotic spindle. Isolated cDNAs for the antigen encode a 622-amino acid polypeptide. Sequence analysis revealed the presence of 340-amino acid residues in the COOH terminus, which is homologous to the **motor** domain conserved among other members of the **kinesin** superfamily. The protein is composed of a central alpha-helical portion with globular domains at both NH2 and COOH termini, and the epitope to the monoclonal antibody resides in the central alpha-helical stalk. A series of deletion constructs were created for in vitro analysis of microtubule interactions. While the microtubule binding and bundling activities require both the presence of the COOH terminus and the alpha-helical domain, the NH2-terminal half of the antigen lacked the ability to interact with microtubules. The full-length as well as deleted proteins consisting of the COOH-terminal **motor** and the central alpha-helical stalk supported microtubule gliding, with velocity ranging from 1.0 to 8.4 mu m/minute. The speed of microtubule movement decreased with decreasing lengths of the central stalk attached to the COOH-terminal **motor**. The microtubules moved with their plus end leading, indicating that the antigen is a minus end-directed **motor**. The CHO2 sequence shows 86% identify to HSET, a gene located at the centromeric end of the **human** MHC region in chromosome 6 (Ando, A., Y. Y. Kikuti, H. Kawata, N. Okamoto, T. Imai, T. Eki, K. Yokoyama, E. Soeda, T. Ikemura, K. Abe, and H. Inoko. 1994. Immunogenetics. 39:194-200), indicating that HSET might represent a **human** homologue of the CHO2 antigen.

L20 ANSWER 28 OF 34 MEDLINE on STN

ACCESSION NUMBER: 95196755 MEDLINE

DOCUMENT NUMBER: PubMed ID: 7889940

TITLE: Mitotic HeLa cells contain a **CENP-E** -associated minus end-directed microtubule **motor**.

AUTHOR: Thrower D A; Jordan M A; Schaar B T; Yen T J; Wilson L

CORPORATE SOURCE: Department of Biological Sciences, University of California, Santa Barbara 93106.

CONTRACT NUMBER: CA06927 (NCI)
GM44762 (NIGMS)

SOURCE: EMBO journal, (1995 Mar 1) 14 (5) 918-26.
Journal code: 8208664. ISSN: 0261-4189.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199504

ENTRY DATE: Entered STN: 19950427

Last Updated on STN: 19970203
Entered Medline: 19950420

AB A minus end-directed microtubule **motor** activity from extracts of HeLa cells blocked at prometaphase/metaphase of mitosis with vinblastine has been partially purified and characterized. The **motor** activity was eliminated by immunodepletion of Centromere binding protein E (**CENP-E**). The **CENP-E**-associated **motor** activity, which was not detectable in interphase cells, moved microtubules at mean rates of 0.46 micron/s at 37 degrees C and 0.24 micron/s at 25 degrees C. The **motor** activity co-purified with **CENP-E** through several purification procedures. **Motor** activity was clearly not due to dynein or to **kinesin**. The microtubule gliding rates of the **CENP-E**-associated **motor** were different from those of dynein and **kinesin**. In addition, the pattern of nucleotide substrate utilization by the **CENP-E**-associated **motor** and the sensitivity to inhibitors were different from those of dynein and **kinesin**. The **CENP-E**-associated **motor** had an apparent native molecular weight of 874,000 Da and estimated dimensions of 2 nm x 80 nm. This is the first demonstration of **motor** activity associated with **CENP-E**, strongly supporting the hypothesis that **CENP-E** may act as a minus end-directed microtubule **motor** during mitosis.

L20 ANSWER 29 OF 34 MEDLINE on STN
ACCESSION NUMBER: 95122643 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7822426
TITLE: Identification and partial characterization of mitotic centromere-associated **kinesin**, a **kinesin**-related protein that associates with centromeres during mitosis.
COMMENT: Comment in: J Cell Biol. 1995 Jan;128(1-2):1-4. PubMed ID: 7822407
AUTHOR: Wordeman L; Mitchison T J
CORPORATE SOURCE: Department of Physiology and Biophysics, University of Washington, Seattle 98195.
CONTRACT NUMBER: CA-09270 (NCI)
GM-39565 (NIGMS)
SOURCE: Journal of cell biology, (1995 Jan) 128 (1-2) 95-104.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950223
Last Updated on STN: 20021227
Entered Medline: 19950214

AB Using antipeptide antibodies to conserved regions of the **kinesin** **motor** domain, we cloned a **kinesin**-related protein that associates with the centromere region of mitotic chromosomes. We call the protein MCAK, for mitotic centromere-associated **kinesin**. MCAK appears concentrated on centromeres at prophase and persists until telophase, after which time the localization disperses. It is found throughout the centromere region and between the kinetochore plates of isolated mitotic CHO chromosomes, in contrast to two other kinetochore-associated microtubule **motors**: cytoplasmic dynein and **CENP-E** (Yen et al., 1992), which are closer to the outer surface of the kinetochore plates. Sequence analysis shows MCAK to be a **kinesin**-related protein with the **motor** domain located in the center of the protein. It is 60-70% similar to kif2, a **kinesin**-related protein originally cloned from mouse brain with a centrally located **motor** domain (Aizawa et al., 1992). MCAK protein is present in interphase and mitotic CHO cells and is transcribed

as a single 3.4-kb message.

L20 ANSWER 30 OF 34 SCISEARCH COPYRIGHT (c) 2004 The Thomson Corporation. on STN
ACCESSION NUMBER: 95:65950 SCISEARCH
THE GENUINE ARTICLE: QB175
TITLE: HETEROGENEITY AND MICROTUBULE INTERACTION OF THE CHO1 ANTIGEN, A MITOSIS-SPECIFIC **KINESIN**-LIKE PROTEIN - ANALYSIS OF SUBDOMAINS EXPRESSED IN INSECT SF9 CELLS
AUTHOR: KURIYAMA R (Reprint); DRAGASGRANOIC S; MAEKAWA T; VASSILEV A; KHODJAKOV A; KOBAYASHI H
CORPORATE SOURCE: UNIV MINNESOTA, DEPT CELL BIOL & NEUROANAT, 4-135 JACKSON HALL, 321 CHURCH ST SE, MINNEAPOLIS, MN, 55455 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF CELL SCIENCE, (DEC 1994) Vol. 107, Part 12, pp. 3485-3499.
ISSN: 0021-9533.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 52
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The CHO1 antigen is a mitosis-specific **kinesin**-like **motor** located at the interzonal region of the spindle. The **human** cDNA coding for the antigen contains a domain with sequence similarity to the **motor** domain of **kinesin**-like protein (Nislow et al., Nature 359, 543, 1992). Here we cloned cDNAs encoding the CHO1 antigen by immunoscreening of a CHO Uni-Zap expression library, the same species in which the original monoclonal antibody was raised, cDNAs of CHO cells encode a 953 amino acid polypeptide with a calculated molecular mass of 109 kDa. The N-terminal 73% of the antigen was 87% identical to the **human** clone, whereas the remaining 27% of the coding region showed only 48% homology. Insect Sf9 cells infected with baculovirus containing the full-length insert produced 105 and 95 kDa polypeptides, the same doublet identified as the original antigen in CHO cells. Truncated polypeptides corresponding to the N-terminal **motor** and C-terminal tail produced a 56 and 54 kDa polypeptide in Sf9 cells, respectively. Full and N-terminal proteins co-sedimented with, and caused bundling of, brain microtubules in vitro, whereas the C-terminal polypeptide did not. Cells expressing the N terminus formed one or more cytoplasmic processes. Immunofluorescence as well as electron microscopic observations revealed the presence of thick bundles of microtubules, which were closely packed, forming a marginal ring just beneath the cell membrane and a core in the processes. The diffusion coefficient and sedimentation coefficient were determined for the native CHO1 antigen by gel filtration and sucrose density gradient centrifugation, respectively. The native molecular mass of overinduced protein in Sf9 cells was calculated as 219 kDa, suggesting that the antigen exists as a dimer. Intrinsic CHO1 antigen in cultured mammalian cells forms a larger native complex (native molecular mass, 362 kDa), which may suggest the presence of additional molecule(s) associating with the CHO1 **motor** molecule.

L20 ANSWER 31 OF 34 MEDLINE on STN
ACCESSION NUMBER: 94266962 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8207059
TITLE: Cyclin-like accumulation and loss of the putative kinetochore **motor CENP-E** results from coupling continuous synthesis with specific degradation at the end of mitosis.
AUTHOR: Brown K D; Coulson R M; Yen T J; Cleveland D W
CORPORATE SOURCE: Department of Biological Chemistry, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205.
CONTRACT NUMBER: GM 29513 (NIGMS)

SOURCE: Journal of cell biology, (1994 Jun) 125 (6) 1303-12.
Journal code: 0375356. ISSN: 0021-9525.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199407
ENTRY DATE: Entered STN: 19940721
Last Updated on STN: 19970203
Entered Medline: 19940712

AB **CENP-E** is a **kinesin**-like protein that binds to kinetochores through the early stages of mitosis, but after initiation of anaphase, it relocalizes to the overlapping microtubules in the midzone, ultimately concentration in the developing midbody. By immunoblotting of cells separated at various positions in the cell cycle using centrifugal elutriation, we show that **CENP-E** levels increase progressively across the cycle peaking at approximately 22,000 molecules/cell early in mitosis, followed by an abrupt (> 10 fold) loss at the end of mitosis. Pulse-labeling with [35S]methionine reveals that beyond a twofold increase in synthesis between G1 and G2, interphase accumulation results primarily from stabilization of **CENP-E** during S and G2. Despite localizing in the midbody during normal cell division, **CENP-E** loss at the end of mitosis is independent of cytokinesis, since complete blockage of division with cytochalasin has no affect on **CENP-E** loss at the M/G1 transition. Thus, like mitotic cyclins, **CENP-E** accumulation peaks before cell division, and it is specifically degraded at the end of mitosis. However, **CENP-E** degradation kinetically follows proteolysis of cyclin B in anaphase. Combined with cyclin A destruction before the end of metaphase, degradation of as yet unidentified components at the metaphase/anaphase transition, and cyclin B degradation at or after the anaphase transition, **CENP-E** destruction defines a fourth point in a mitotic cascade of timed proteolysis.

L20 ANSWER 32 OF 34 MEDLINE on STN
ACCESSION NUMBER: 94294810 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8023161
TITLE: Mitotic regulation of microtubule cross-linking activity of **CENP-E** kinetochore protein.
AUTHOR: Liao H; Li G; Yen T J
CORPORATE SOURCE: Fox Chase Cancer Center, Philadelphia, PA 19111.
CONTRACT NUMBER: CA-06927 (NCI)
GM-44762-02 (NIGMS)
SOURCE: Science, (1994 Jul 15) 265 (5170) 394-8.
Journal code: 0404511. ISSN: 0036-8075.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 19940815
Last Updated on STN: 19940815
Entered Medline: 19940802

AB **CENP-E** is a **kinesin**-like protein that is transiently bound to kinetochores during early mitosis, becomes redistributed to the spindle midzone at anaphase, and is degraded after cytokinesis. At anaphase, **CENP-E** may cross-link the interdigitating microtubules in the spindle midzone through a **motor**-like binding site at the amino terminus and a 99-amino acid carboxyl-terminal domain that bound microtubules in a distinct manner. Phosphorylation of the carboxyl terminus by the mitotic kinase maturation promoting factor (MPF) inhibited microtubule-binding activity before anaphase. Thus, MPF suppresses the microtubule cross-linking activity of

CENP-E until anaphase, when its activity is lost.

L20 ANSWER 33 OF 34 MEDLINE on STN
ACCESSION NUMBER: 94168458 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8122906
TITLE: With apologies to scheherazade: tails of 1001 **kinesin motors**.
AUTHOR: Goldstein L S
CORPORATE SOURCE: Department of Cellular and Developmental Biology, Harvard University, Cambridge, Massachusetts 02138.
SOURCE: Annual review of genetics, (1993) 27 319-51. Ref: 98
Journal code: 0117605. ISSN: 0066-4197.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199404
ENTRY DATE: Entered STN: 19940412
Last Updated on STN: 19940412
Entered Medline: 19940404

L20 ANSWER 34 OF 34 MEDLINE on STN
ACCESSION NUMBER: 93024922 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1406971
TITLE: **CENP-E** is a putative kinetochore **motor** that accumulates just before mitosis.
COMMENT: Comment in: Nature. 1992 Oct 8;359(6395):480-2. PubMed ID: 1406965
AUTHOR: Yen T J; Li G; Schaar B T; Szilak I; Cleveland D W
CORPORATE SOURCE: Fox Chase Cancer Center, Philadelphia, Pennsylvania 19111.
SOURCE: Nature, (1992 Oct 8) 359 (6395) 536-9.
Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199211
ENTRY DATE: Entered STN: 19930122
Last Updated on STN: 19930122
Entered Medline: 19921113

AB The mechanics of chromosome movement, mitotic spindle assembly and spindle elongation have long been central questions of cell biology. After attachment in prometaphase of a microtubule from one pole, duplicated chromosome pairs travel towards the pole in a rapid but discontinuous motion. This is followed by a slower congression towards the midplate as the chromosome pair orients with each kinetochore attached to the microtubules from the nearest pole. The pairs disjoin at anaphase and translocate to opposite poles and the interpolar distance increases. Here we identify **CENP-E** as a **kinesin**-like **motor** protein (M(r) 312,000) that accumulates in the G2 phase of the cell cycle. **CENP-E** associates with kinetochores during congression, relocates to the spindle midzone at anaphase, and is quantitatively discarded at the end of the cell division. **CENP-E** is likely to be one of the **motors** responsible for mammalian chromosome movement and/or spindle elongation.

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(FILE 'HOME' ENTERED AT 08:59:27 ON 17 SEP 2004)

FILE 'MEDLINE, EMBASE, BIOSIS, BIOTECHDS, SCISEARCH, HCAPLUS, NTIS,

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     LIFESCI' ENTERED AT 08:59:47 ON 17 SEP 2004
L1         14477 S KINESIN?
L2           831 S "CENP-E"
L3             1 S "CENTROMER BINDING"
L4             0 S CENTROMER (2W) "PROTEIN E"
L5           282 S L1 AND L2
L6           125 S HUMAN AND L5
L7            67 S MOTOR AND L6
L8        333307 S ATPASE
L9             6 S L6 AND L8
L10            6 DUP REM L9 (0 DUPLICATES REMOVED)
L11           30 DUP REM L7 (37 DUPLICATES REMOVED)
                 E BEARUD C/AU
                 E BERAUD C/AU
L12          478 S E3
                 E OHASHI C/AU
L13           26 S E3
                 E SAKOWICZ R/AU
L14           76 S E5
                 E VAISBERG E/AU
L15           30 S E3
                 E WOOD K/AU
L16          803 S E3
                 E YU M/AU
L17         2350 S E3
L18         3786 S L11 OR L12 OR L13 OR L14 OR L15 OR L16 OR L17
L19           36 S L2 AND L18
L20           34 DUP REM L19 (2 DUPLICATES REMOVED)
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	Issue Date	Pages	Document ID	Title
1	20040722	27	US 20040142397 A1	Novel motor proteins and methods for their use
2	20040429	27	US 20040081982 A1	Neocentromere-based mini-chromosomes or artificial chromosomes
3	20031218	42	US 20030232832 A1	Pyrrolotriazinone compounds and their use to teat diseases
4	20030501	43	US 20030083261 A1	Class of 12mer peptides that inhibit the function of the mitotic check point protein Mad2
5	20030306	19	US 20030044900 A1	Human kinesins and methods of producing and purifying human kinesins
6	20030109	32	US 20030008888 A1	Novel cyano-substituted dihydropyrimidine compounds and their use to treat diseases
7	20021107	18	US 20020165240 A1	Method of treating proliferative diseases using Eg5 inhibitors
8	20021003	37	US 20020143026 A1	Cyano-substituted dihydropyrimidine compounds and their use to treat diseases
9	20020214	22	US 20020019704 A1	Significance analysis of microarrays
10	20040720	27	US 6764830 B1	Thermomyces lanuginosus kinesin motor protein and methods of screening for modulators of kinesin proteins
11	20040713	26	US 6762043 B1	Motor proteins and methods for their use
12	20040615	24	US 6750330 B1	Lyophilized tubulins

	Issue Date	Pages	Document ID	Title
13	20040420	28	US 6723840 B1	Identification and expression of a novel kinesin motor protein
14	20031111	38	US 6645748 B1	Plus end-directed microtubule motor required for chromosome congression
15	20030715	46	US 6593098 B1	Genes encoding proteins involved in mitotic checkpoint control and methods of use thereof
16	20020514	26	US 6387644 B1	Motor proteins and methods for their use
17	19980120	43	US 5710022 A	Nuclear mitotic phosphoprotein

	Issue Date	Pages	Document ID	Title
1	20040909	29	US 20040176625 A1	Kinesin motor modulators derived from the marine sponge Adocia
2	20030710	30	US 20030127621 A1	Kinesin motor modulators derived from the marine sponge adocia
3	20040817	31	US 6777200 B2	Kinesin motor modulators derived from the marine sponge Adocia
4	20031111	38	US 6645748 B1	Plus end-directed microtubule motor required for chromosome congression
5	20021203	24	US 6489134 B1	Kinesin motor modulators derived from the marine sponge Adocia
6	20010327	26	US 6207403 B1	Kinesin motor modulators derived from the marine sponge Adocia

	Issue Date	Pages	Document ID	Title
1	20040909	29	US 20040176625 A1	Kinesin motor modulators derived from the marine sponge Adocia
2	20040722	27	US 20040142397 A1	Novel motor proteins and methods for their use
3	20040318	617	US 20040052820 A1	Fusion proteins comprising DP-178 and other viral fusion inhibitor peptides useful for treating aids
4	20040304	107	US 20040044184 A1	Cytoskeleton-associated proteins
5	20040304	95	US 20040043037 A1	Staphylococcus aureus polynucleotides and sequences
6	20040219	889	US 20040033235 A1	Nucleic acids encoding DP-178 and other viral fusion inhibitor peptides useful for treating aids
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11	20030501	43	US 20030083261 A1	Class of 12mer peptides that inhibit the function of the mitotic check point protein Mad2
12	20030320	109	US 20030054436 A1	STAPHYLOCOCCUS AUREUS POLYNUCLEOTIDES AND SEQUENCES
13	20030306	19	US 20030044900 A1	Human kinesins and methods of producing and purifying human kinesins
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18	20040720	27	US 6764830 B1	Thermomyces lanuginosus kinesin motor protein and methods of screening for modulators of kinesin proteins
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25	20030715	97	US 6593114 B1	Staphylococcus aureus polynucleotides and sequences
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30	20021203	24	US 6489134 B1	Kinesin motor modulators derived from the marine sponge Adocia
31	20021126		US 6485925 B1	Anthrax lethal factor is a MAPK kinase protease
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35	20020219		US 6348353 B1	Artificial mammalian chromosome
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